A PROSPECTIVE STUDY ON CLINICAL PROFILE AND MANAGEMENT OF ACUTE UPPER GASTRO INTESTINAL BLEEDING AMONG 50 CASES IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM

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ACUTE UPPER GASTRO INTESTINAL BLEEDING

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation entitled "**A PROSPECTIVE STUDY ON CLINICAL PROFILE AND MANAGEMENT OF ACUTE UPPER GASTRO INTESTINAL BLEEDING AMONG 50 CASES IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM**" is a bonafide work done by DR. MOHAMMED MINNATHULLAH, S. A. under my guidance during the period of 2011-2013. This has been submitted to the partial fulfilment of the award of M.S. degree in General Surgery (Branch I) Tamil Nadu Dr. M.G.R. Medical University, Chennai-32.

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DECLARATION

I solemnly declare that this dissertation "**A PROSPECTIVE STUDY ON CLINICAL PROFILE AND MANAGEMENT OF ACUTE UPPER GASTRO INTESTINAL BLEEDING AMONG 50 CASES IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM**" was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem-636030 under the guidance and supervision of PROF. DR. R. KATTABOMMAN, M.S., HOD of General Surgery, Govt. Mohan Kumaramangalam Medical College and Hospital, Salem. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in fulfilment of the University regulations for the award of the degree of M.S. General Surgery (Branch I).

Place: Salem

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DR. MOHAMMED MINNATHULLAH, S. A.

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LIST OF ABBREVIATIONS USED

UGIB –Upper Gastro Intestinal Bleed

GI – Gastro Intestinal

LGA – Left Gastric Artery

GEA – Gastro Epiploic Artery

SGA – Short Gastric Artery

INR – International Normalised Ratio

HPVG – Hepatic Venous Pressure Gradient

N – Normal

SIGN – Scottish Inter Collegiate Guidelines Network

M – Male

F – Female

U – Urban

R – Rural

PR – Pulse Rate

BP – Blood Pressure

PCV – Packed Cell Volume

RAS – Rock All Score

ATLS – Advanced Trauma Life Support

PPI – Proton Pump Inhibitors

S – Sclerotherapy

B – Banding

NGA – Naso Gastric Aspirate

R-HUN – Right Hydro Urethro Nephrosis

B – Brown

R – Red

C – Clear

D – Discharged

E – Expired

ABSTRACT

Background

Upper GI Bleeding is a medical and surgical mortality rate accounts to 10%. Most of the patients presents to hospital in hypovolemia and shock. Considering all these, this study on 50 patients presented with upper GI bleeding admitted in our hospital, GMKMCH, Salem enabled us to identify commonest etiology, clinical presentation and prognosis in the locality.

Methodology

The cases were evaluated through proper history taking, thorough clinical examination, endoscopic examination and were grouped under seven groups and were analyzed. Patients less than 15 years and patients not stabilized within 48 hours of resuscitation were excluded from the study.

Observation

Duodenal ulcer was the commonest cause of upper GI bleed (37%) followed by esophageal varices (20%). Peak incidence was among 35 – 45 years with male predominance. Most patients presented with both hematemesis and malena (52%). Patients were treated with PPIs, sclerotherapy (10) or banding (9).

Conclusion

Peptic ulcer is the commonest cause. Sex and blood group distribution were similar to other studies. Death due to upper GI bleed is still a challenge for the investigators too.

INTRODUCTION

Upper GI Bleeding is a medical and surgical emergency. Most commonly patients presents with hematemesis and malena. [1] Upper GI Bleeding denotes bleeding above the ligament of Treitz. Malena is foul smelling tarry stools due to altered blood. [2] 50 to 100 ml of blood loss is required to produce malena.

Mortality from bleeding increases with age. [3] Overall mortality accounts to 10%. [2] Mortality in less than 60 years without co-morbidities like malignancy or organ failure accounts to less than 1%. Mortality from first bleed from varices is 50%. [4] Inpatient mortality due to rebleeding amounts to 30%. [5]

The best investigation in upper GI bleed is upper GI endoscopy. It enables diagnosis appropriate therapy and prognostic significance. [5]

With diverse etiology and fatal outcome all cases of recent GI bleed should be admitted and evaluated and treated accordingly. When patient is in shock or when haemoglobin is less than 10g/ml, patient should be stabilized and subjected for OGD scopy to identify the cause for prompt management.

Considering all these factors, this study on 50 patients presented with upper GI bleed admitted in our hospital, GMKMCH, Salem, enabled to identify most common etiology clinical presentation and prognosis in the locality and will

help create awareness among all levels of Medical practitioners and enhance prompt diagnosis and at least early referral.

This helps reducing suffering and cost of expensive investigations for the poor patients in this locality.

AIM OF THE STUDY

1. To assess the clinical status of the patient getting admitted.

2. Work up including OGD Scopy.

3. To analyse the cause.

4. To analyse the different Management modalities.

5. To find the period of hospital stay.

6. To analyse the outcome.

REVIEW OF LITERATURE

HISTORICAL REVIEW

1. **Vaclav Treitz** first described the "**Ligament of Treitz**" also known as suspensory muscle of duodenum in 1853.

2. **Ryles tube** was first introduced in clinical practice by **Abraham Louis Levin** in 1927

3. **Helico bacter pylori** as a cause of peptic ulcer was first identified in1983 by **Dr.barry J.Marshall and Dr.J.Robin warren** at Perth

4. **SengstakenBlakemore** tube was introduced by **Robert Sengstaken and Arthur Hendley Blakemore** in 1950

5. **Octreotide** first synthesized in 1979 by chemist **Wilfried Bauer**

6. **Mallory Weiss** tear first identified by **G.Kenneth Mallory and Soma Weiss in 1929**

7. **Rockall scoring system** was introduced by **Prof.Tim Rockall** in 1996

8. **Basil Hischowitz and Larry Curtiss** invented first fibre optic endoscope in **1957**

9. **Sclerotherapy** was first introduced by **D.Zollikofer in** Switzerland in 1982

10. **Banding** techniques was first introduced by **Wilkinson and peloso in 1978**

11. **TIPS** procedure was first described by **Josef Rosch in 1969**

ANATOMY OF STOMACH

The stomach is the first intra-abdominal part of the gastrointestinal (GI), or digestive, tract. It is a muscular, highly vascular. The cardiac notch (incisura cardiaca gastri) is the acute angle between the abdominal esophagus and the fundus of the stomach, which is the part of stomach above a horizontal line drawn from the cardia. The body (corpus) of the stomach leads to the pyloric antrum (at the incisura angularis). The pyloric antrum narrows toward the right to become the pyloric canal, surrounded by the pyloric sphincter, which joins the duodenum at the L1 level (transpyloric plane) to the right of the midline.

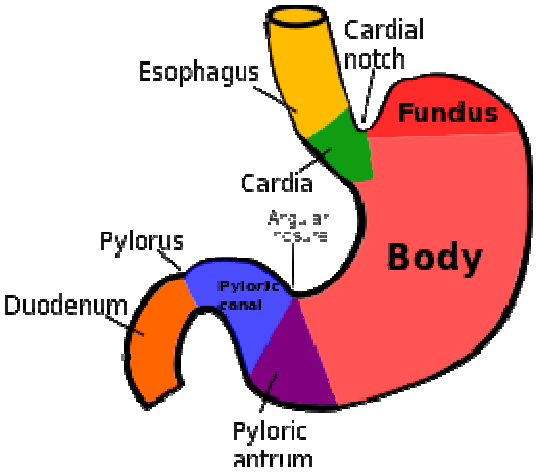


FIG 1.PARTS OF STOMACH

The celiac trunk (axis) arises from the anterior surface of the abdominal aorta at the level of L1. It has a short length (about 1 cm) and trifurcates into the

common hepatic artery (CHA), the splenic artery, and the left gastric artery (LGA).

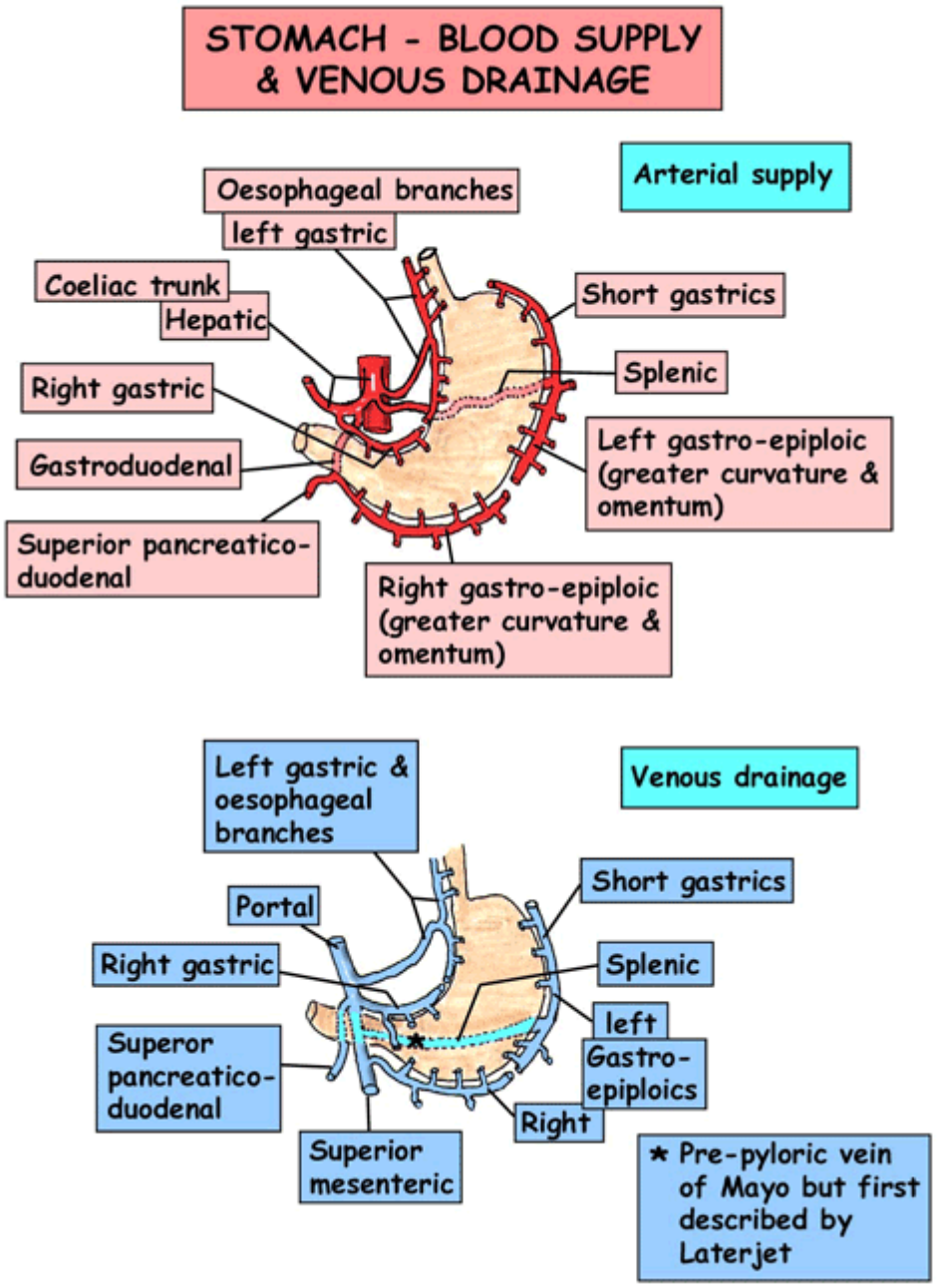


FIG 2: BLOOD SUPPLY OF STOMACH

The LGA runs toward the lesser curvature of the stomach and divides into an ascending branch (supplying the abdominal esophagus) and a descending

branch (supplying the stomach). The CHA runs toward the right on the superior border of the pancreas and gives off the gastroduodenal artery (GDA), which runs down behind the first part of the duodenum. After giving off the GDA, the CHA continues as the proper hepatic artery.

The right gastric artery, a branch from the proper hepatic artery, travels along the lesser curvature from right to left and joins the descending branch of the LGA to form an arcade along the lesser curvature between the 2 leaves of peritoneum of the lesser omentum. This arcade gives off multiple small arteries to the body of the stomach. The GDA divides into the right gastro-omental (gastroepiploic) artery (RGEA) and the anterior superior pancreaticoduodenal artery (SPDA); it also gives off the small supraduodenal artery (of Wilkie). The RGEA runs along the greater curvature from right to left.

The splenic artery runs toward the left on the superior border of the distal body and tail of pancreas and gives off the left gastro-omental artery (LGEA), which runs from left to right along the greater curvature and joins the RGEA to form an arcade along the greater curvature between the two leaves of peritoneum of the greater omentum.

The greater curvature arcade formed by the RGEA and the LGEA provides several omental (epiploic) branches to supply the highly vascular greater

omentum. The splenic artery also gives off 3-5 short gastric arteries that run in the gastrosplenic ligament and supply the upper part of the greater curvature and the gastric fundus. The stomach has a rich network of vessels in its submucosa. The left gastric (coronary) vein drains into the portal vein at its formation (by the union of the splenic and superior mesenteric veins). The right gastric and right gastro-omental veins drain into the portal vein. The left gastro-omental vein drains into the splenic vein, as do the short gastric veins.

The pylorus is marked by an angular or prepyloric vein (of Mayo), which lies along the incisura angularis. The gastrocolic trunk is present in a large number of cases and lies at the junction of the small bowel mesentery and the transverse mesocolon. It may drain the right colic, middle colic, and right gastro-omental veins.

The short gastric arteries and veins are sometimes collectively referred to as the vasa brevia.

KNOWLEDGE ABOUT UPPER GI BLEEDING

Haemorrhage

Most patients with hematemesis and malena present in the hospital as primary bleeder while some patients (secondary bleeders) who are critically ill develop

hematemesis and malena in the hospital. The mortality is very high in patient with bleeding that develops in the hospital and this is usually a result of systemic diseases. (6)

Hematemesis means the vomiting of fresh blood, blood with clots, or blood which has been subjected to digestion by gastric juices.

Malena is the term used to describe passage of black tarry stool containing altered blood; this is usually due to bleeding from upper GI tract, although haemorrhage from the right side of the colon is occasionally responsible.
Stools are tarrying ("sticky") and have a characteristic odour. (7)

Factors responsible for tarry stools are:

1. Site of bleeding
2. Amount of bleeding
3. Rapidity of bleeding
4. Intestinal transit time
5. Bacterial breakdown of haemoglobin in the intestine.

Haematochezia is the passage of bright red blood per rectum, generally signifies bleeding from a source distal to the ligament of treitz. However, brisk proximal bleeding can cause haematochezia due to rapid transit.

ETIOLOGY OF UPPER GI BLEED:

Most common presentation is hematemesis and malena. Common causes include:

1. Peptic Ulcer - Duodenal Ulcer And Gastric Ulcer
2. Erosive Gastritis
3. Variceal Bleed – Portal Hypertention
4. Reflux Esophagitis
5. Mallory – Weiss Tear
6. Vascular Malformation

Uncommon causes include:

1. Gastric carcinoma-lymphoma, polyps
2. Duodenal or jejunal diverticuli
3. Aorto duodenal fistulas
4. Primary aorto esophageal fistula
5. Blood dyscrasias
6. Vasculitis
7. Hemorrhagic telangiectasia

8. Connective tissue disorder
9. Uremia
10. Undoubtful sources

In a study conducted in 1534 patients in Greece with upper GI bleeding, it was found that peptic ulcer (67%) is the most common cause. [8]

In a study conducted in 5000 patients in Thailand, it was evident that peptic ulcer contributed 51.24%.

In National American Society of Gastroenterology (ASGE) survey on upper GIT on 2225 patients, duodenal & gastric ulcer were the commonest cause of upper GI bleeding. [9]

In recent years, UGI bleeding has been associated with the ingestion of NSAIDs for arthritis about 35% patients with GI bleeding gives history of consuming NSAIDSs a week prior to presentation. Majority of them were above 60 years. [10]

In a study in Somerville K^{20} shows that 35-55% of elderly patients admitted with upper GI bleeding has taken NSAIDS with 10% mortality. Vascular anamolies like vascular ectasias contribute to 7%.

PATHOPHYSIOLOGY

Variety of factors contributes to the development of peptic ulcer disease. Final common pathway to ulcer formation is acid – peptic injury of gastroduodenal mucosal barrier. The adage “No acid - No ulcer” remains true even today. (11)

Duodenal ulcer has typically been viewed as a disease of increased acid peptic action on the duodenal mucosa, whereas gastric ulcer disease of weakened mucosal defenses even with decreased acid – peptic activity.

Peptic ulcer mainly due to imbalance between the aggression, defence and repair factors. (13)

A. Aggression Factors
    i. Acid
    ii. Pepsin
    iii. NSAIDS
    iv. H.pylori

B. Defense Factors
    i. Bicarbonate
    ii. Blood flow

iii. Mucous

iv. Cell junction

v. Apical resistance

C. Repair Mechanism

i. Restitution

ii. Mucoid Cap

iii. Proliferation

iv. Growth Factors

Acid Secretion and Peptic Ulcer

Duodenal Ulcer patients have higher mean basal acid output (BAO) and higher mean acid output (MAO) compared to normal controls. Nocturnal acid secretion in increased than day time secretion. The patients have exaggerated response to any gastric acid stimulus. The patients also have increased parietal cell response to gastrin. (11)

Factors responsible for duodenal ulcers are: (12)

i. Increased Basal Acid Output

ii. Increased Gastric acid response to gastrin

iii. Impaired feedback mechanism

iv. Increased rate of gastric emptying

v. Increased acid load to the duodenum at any given time

vi. Decreased duodenal bicarbonate secretion and buffering capacity

Factors responsible for gastric ulcers are:

i. Weak mucosal defenses

ii. Duodeno gastric reflex

iii. NSAIDS

iv. H.pylori

v. Chronic gastritis

vi. Aspirin

Gastric ulcers are due to weak mucosal defenses that permit abnormal amount of injurious acid, back diffusion into the mucosa. Duodeno gastric reflex causes reflex of bile, lysolecithin, pancreatic juice which are injurious to gastric mucosa.

Types of Gastric Ulcer

Johnsons Classification: [11]

Type 1 – ulcer near angularis incisura with normal or decreased acid production

Type II – active or quiescent duodenal ulcer with normal or increased acid secretion

Type III – prepyloric ulcer with normal or increased acid secretion

Type IV – ulcer at gastroesophageal junction with normal or decreased acid secretion

Type V – ulcer anywhere associated with medications

Helicobacter Pylori and Peptic Ulcer Disease

Patients with H.Pylori induced antral gastritis are 3 ½ times more likely to develop PUD than the patients without H.Pylori infection. 90% of Duodenal Ulcer and 70%-90%of Gastric Ulcer has H.Pylori infection. It causes hyper gastrenemia and acid hyper secretion which results in antral gastritis and antral epithelial metaplasia in post Pyloric duodenum.

NSAIDS in Peptic Ulcer Disease

Patients with rheumatoid arthritis or osteoarthritis on NSAIDS have 15-20% annual incidents of peptic ulcer disease. Prevalence of peptic ulcer disease in chronic NSAID users is 25%. There is an extra 5% increase in risk in patients above 60 years.

Factors that increase the risk of NSAIDS induced peptic ulcer disease are:

1) Age more than 60 years
2) Prior gastro intestinal events
3) High NSAIDS dosage
4) Concurrent steroid intake
5) Concurrent anticoagulant therapy

Smoking Induced Peptic Ulcer

Smokers are about twice as likely to develop PUD when compared to non-smokers because smoking causes[12]

1) Increased gastric acid secretion
2) Duodeno gastric reflux
3) Decreases gastroduodenal prostaglandin production
4) Decreases pancreatico duodenal bicarbonate production

Alcohol and Peptic Ulcer Disease [11]

Alcohol is being commonly mentioned as a risk factor. Confirmatory data are lacking. Hudson proposed possibility of alcohol causing erosive mucosal disease.

Stress and Peptic Ulcer [12]

Cushing described acute peptic ulcer in patients in head trauma.

Curlings described duodenal ulcer/duodenitis in burns patients.

Other causes of Peptic Ulcer disease includes [14]

1) Gastrinoma / MEN Type 1 Syndrome
2) Chronic Obstructive Pulmonary Disease
3) Chronic Renal Failure
4) Cirrhosis Liver
5) Systemic Mastocytosis

Erosive gastritis [12]

It denotes mucosal inflammation. Erosion is mere shedding of superficial epithelium of stomach and occasionally of duodenum. The causes include: [12]

1) H.Pylori
2) Alcohol
3) NSAIDS
4) Crohns Disease
5) Tuberculosis
6) Bile reflux

The above factors cause mucosal damage by immune cell infiltration and cytokine production. Alchohol, aspirin and bile additionally causes mucosal damage by back diffusion of Luminal hydrogen ions.

Stress Induced Gastritis [12]

Gastritis due to stress is on the decline now due to better critical care with acid suppression and cytoprotective drugs. Stress induced gastritis caused due to inadequate gastric mucosal blood flow. Causes include:

1) Shock
2) Burns
3) Sepsis
4) Severe trauma and head injury

Features of Gastric Erosion [15]

1) Ulcer less than 1 cm diameter
2) Rarely penetrates mucosa.
3) Absence of scarring or thickening of blood vessels
4) Heals with complete epithelisation

Recurrent ulcer [16]

Recurrent ulcer occurs in approximately 5% of all patients after surgery for peptic ulcer. Risk of recurrence is 3 to 10% after surgery for duodenal ulcer and 2% after gastric ulcer surgery. Causes include:

1) Inadequate surgical procedure
2) Zollinger Ellison syndrome
3) Gastric/duodenal carcinoma
4) Persistent H.pylori infection
5) Non compliant patient to drugs
6) Motility disorder

Isolated gastric varices [12]

It occurs in absence of esophageal varices. It includes two types.

Type 1- Fundic Varices

Type 2- Distal to Fundus (including proximal duodenum)

The commonest causes include portal hypertension and splenic vein thrombosis.

Mallory Weiss Tear [12]

Mallory Weiss Tear is a longitudinal tear in the mucosa of the gastro esophageal junction presumably caused by forceful vomiting or retching. It is commonly seen in alcoholics. Patients present with hematemesis. 90% of patients stop bleeding spontaneously.

Esophageal varices

Varices are tortuous veins within the submucosa of distal esophagus and proximal stomach. Esophageal varices are mainly supplied by left gastric vein and gastric varices are mainly supplied by short gastric veins. [17]

Bleeding from varices may occur when portal pressure exceeds 11 to 12 mm of Hg above the IVC pressure. Esophageal varices are a common cause of bleeding with a mortality of 50%. [18] Most survivors rebleed with an impatient mortality of 30%.

Watermelon Stomach [12]

It is a gastric antral vascular ectasia. It is characterized by dilated mucosal blood vessels in the lamina propria. It is associated with mucosal fibro muscular

hyperplasia and hyalinization. It differs from portal hypertensive gastropathy by preferentially affecting the distal stomach.

Dieulafoy's Lesions [(11)]

Congenital arterio venous malformation is characterized by unusual large tortuous submucosal arteries. If these arteries get eroded pulsatile bleeding may occur. It typically occurs in middle aged and elderly men. Patients present with upper GI bleeding. Intermittent endoscopy can miss the lesion.

Investigations:

1. Blood grouping and typing for purposes of blood transfusion if any

2. Haemoglobin Percentage:

- Initial haemoglobin will be baseline because patient is losing whole blood.
- Later due to influence of extra vascular fluid and fluid for resuscitation haemoglobin declines as blood dilutes. Blood haemoglobin is to be monitored every six hours.

3. Total count increases after haemorrhage.

4. Neutrophilic picture in the differential count

5. Platelet count increases after haemorrhage

6. Bleeding, clotting and prothrombin times are assessed to rule out blood dyscrsias.

7. Blood urea, nitrogen increased in upper GI bleed as blood gets absorbed as it passes down the GIT and also as a result of impaired renal perfusion.

8. Liver Function Test

Bilirubin and liver enzymes like SGOT, SGPT are raised in case of liver disease as a cause of Upper GI Bleed.

Prothrombin time is increased in chronic liver disease and in patients on oral anti coagulants.

9. Viral markers. HBsAg, Anti HVC, HBcAg

10. Ultra Sonogram Abdomen

- To assess the hepatic echo texture in case of chronic liver disease and to study portal vein.

11. Nasogastric Aspiration

- To assess whether a patient has ongoing bleeding or not and might be benefitted from an early endoscopy.
- Nasogastric lavage helps to remove clots, blood, and debris from stomach to aid endoscopy and also to assess the rapidity of bleeding.

UPPER GI ENDOSCOPY

Upper GI Endoscopy is the diagnostic modality of choice for an acute upper GI bleeding. [19,44]

Endoscopy has a high sensitivity and specificity for lesion identification. It has both diagnostic and therapeutic advantage by identifying the site of bleeding and ensuring haemostasis. [45]

Early endoscopy (i.e.) within 24 hours is recommended for acute upper GI bleeding which delineates.

a. Site of bleeding
b. Rapidity of bleeding (i.e.) spurting or oozing
c. Any stigmata of recent haemorrhage
d. Assessing associated lesions

Indications:

1. Any patients with acute upper GI bleeding
2. To assess the site of bleeding
3. When surgery is contemplated
4. Rebleeding
5. Therapeutic purpose. Sclerotherapy, banding, electro coagulation

Contra Indications for Upper GI endoscopy

1. Patient non cooperativeness
2. Shock
3. Zenker's diverticulum
4. Comatosed patients
5. Suspected perforation
6. Cardiac decompensation
7. Acute rmyocardial infarction
8. Previous oro pharyngeal surgery (or) radiation therapy
9. Bleeding dyscrasias
10. Poor general condition

Complications

1. Perforation
2. Cardio vascular decompromise

3. Aspiration pneumonitis
4. Bleeding
5. Infection

RISK STRATIFICATION

Several investigations have developed decision and predictive models that permit identifications of patients at low risk of recurrent haemorrhage. Such patients can be discharged at the earliest thereby reducing the resource utilisation prioritizing the cases as risk and delineates patients who require intensive care.

Blotch ford score [20]

Variables	1	2	3	4	5	6
Systolic bp (mmHg)	100-109	90-99	<90	-	-	-
Blood Urea Nitrogen (mg/dl)	-	39.0 -> 47.4	48 – 0 - 59.4	60.0 -> 149.4	-	> 150
Haemoglobin (g/dl)	12 – 12.9	-	10 – 11.9	-	-	< 10
Others	Heart Rate > 100 Malena	Hepatic Disorder Cardiac Failure	-	-	-	-

TABLE 1: Blotch ford scoring SYSTEM IN UPPER GI BLEED.

Scoring system ranges from 0 to 23 and the risk of requiring endoscopic intervention increases with increase in the score. [20]

AIMS 65 SCORE:

It is another scoring system that utilizes data available prior to endoscopy. It has high accuracy for predicting inpatient mortality among patient with upper GI bleeding. [21]

The scoring system includes five factors that are associated with increased inpatients mortality.

i. Albumin less than 3 g/dl
ii. INR > 1.5
iii. Altered mental status (GCS < 14)
iv. Systolic blood pressure ≤ 90 mmHg
v. Age > 65 years

The mortality rate increases as the number of factors increases. It also predicts duration of hospital stay but could not assess rebleeding. [43]

Rockall Score [22]

2008 Scottish Intercollegiate Guidelines Network (SIGN) guideline on the Management of acute upper GI bleed to which recommends that an initial (pre endoscopic) Rockall Score be calculator for all patients presenting with acute UGIB in patients with an initial Rockall Score less than zero.

It is a useful system to access UGIB. The Rockall system is an accurate and valid predictor of rebleeding and death.

It is a summation of point grading system assigned to each of the components bounded on a scale of 0 to 11.

The Rockall Scoring System [23]

	SCORE			
Variable	0	1	2	3
Age (Years)	< 60	60 - 79	≥ 80	
Shock	No Shock Systolic BP > 100 mm Hg HR < 100 bt/min	Tachycardia systolic BP > 100 HR > 100 bt/min	Hypotension systolic BP < 100/min	
Comorbidity	Nil		Cardiac failure Ischemic heart disease or any others.	Renal failure, Liver failure. Disseminated malignancy
Diagnosis	Mallory-Weiss tear No Lesion No SRH	All other diagnosis	Malignancy of Upper GI tract	
Major SRH	None (or) Dark spot		Blood in Upper GI tract adherent clot spurting vessel	

TABLE 2: ROCKALL SCORING SYSTEM.

A score of 0 – 3 is grouped under low risk and 3 – 5 as Moderate Risk and > 5 are high risk groups.

MANAGEMENT OF UPPER GI BLEEDING

Objectives in the Management Of Upper GI Bleeding

- Immediate clinical status assessment
- Stabilize the hemodynamic status
- Identify the source of bleeding
- Stopping the active bleeding
- Treat the underlying cause
- Prevent recurrent bleeding

General Management

All patients with hemodynamic instability (shock, orthostatic hypotension) or active bleeding (i.e.) hematemesis, (or) bright red blood through the naso gastric tube should be admitted in an Intensive Care Unit for resuscitation and close monitoring.

Other patients with upper GI bleed with stable vitals and hemodynamic status and no further bouts of hematemesis can be admitted in routine surgical ward.

General Support

Patient should be kept Nil per oral. Oxygen support given by nasal canula. Two large calibre peripheral intra venous catheter or central venous line should be placed. Elective endo tracheal intubation if patient has altered neurological status which may facilitate, during endoscopy and may prevent aspiration of Gastric contents.

Fluid Resuscitation

Prior to endoscopy, stabilization and resuscitation of the patient is a must to minimize the treatment associated complications.

Crystalloids and colloids administered to restore the blood pressure if hypoalbuminemia is detected, cross match blood and sent blood sample for haemoglobin, haematocrit, BUN. [24]

Monitor urine output, oxygen saturation.

Place patient In trendelenberg position to maintain cerebral blood flow. [25]

Blood Transfusion

The decision to initiate blood transfusions must be individualized.

Haematocrit is maintained around 30%in elderly with associated co-morbidities such as congestive heart failure, chronic renal failure, Ischemic heart disease etc.

Haematocrit to be maintained 20-25% in young patients and about 25-28% in cases with portal hypertension.

However patients with active bleeding and hypovolemia may require blood transfusion despite apparently normal haemoglobin.

Avoid over transfusion in suspected variceal bleeding as it can worse the bleeding.

Patients with active bleeding with prolonged prothrombin time and INR > 1.5 should be corrected with fresh frozen plasma. If INR ≥ 3, it is to be corrected to less than 3 prior endoscopy.[26]

Platelet transfusion is considered if patient has ongoing bleed with platelet count < 50,000 cells/cu.mm and also in patients on oral anti coagulant and anti platelet agents. [27]

Gastric Lavage

It is performed in the view to remove blood clots, within the stomach and to reduce the rate of blood loss. It has been shown that gastric lavage can cause temporary cessation of bleeding in most of patients, regardless of cause of bleeding. Experimental evidence suggests that room temperature tap water lavage is as effective as ice cold saline lavage.

Drug therapy

Acid suppression

Proton Pump Inhibitors

Patients admitted with acute upper GI bleeding are typically treated with proton pump inhibitors (PPI).

High dose anti secretory therapy with an intravenous infusion of a PPI significantly reduces the rate of rebleeding and also decreases the length of hospital stay. [28]

According to 2008 SIGN Guidelines, it recommends that high dose of intravenous PPIs in patient with major peptic ulcer bleeding or non-bleeding visible vessels after endoscopic bleeding control. PPIs have the ability to maintain a gastric Ph at a level above 6.0 and prevents ulcer clot from fibrinolysis.

Lao et al demonstrated high dose of intravenous omeprazole can accelerate the resolution of stigmata of recent hemorrhage and reduce the need for endoscopic therapy.

2010 international consensus guidelines on UGIB recommends use of intravenous PPIs in all patients with high risk lesion post endoscopy theory.

The goal of treatment directed at healing the ulcer and eliminating precipitating factor and also prevents risk of rebleeding.

Diagnostic Approach

History:

UGIB is a medical and surgical emergency which requires early detection and appropriate management. Most common presentation is hematemesis (40 – 50%) and Malena (70 – 80%).

Other history at presentation includes weakness, dizziness, syncope, dyspepsia,sweating, early satiety history of analgesic intake.

History of chronic alcohol intake or chronic hepatitis B or C infection suggests possibility of variceal haemorrhage or portal gastropathy.

History of Epigastric pain and antacid intake related to peptic ulcer disease.

History of dyspepsia, early satiety, NSAID Consumption indicate NSAID gastropathy induced UGIB due to poor platelet aggregation. History of lethargy, cachexia, loss of weight with epigastric discomfort suggest possibility of carcinoma stomach.

History of jaundice, abdominal distention, Pedal edema suggest variceal bleeding due to cirrhosis. Dysphagia and discomfort during swallowing suggest esophageal pathology.

Odynophagia, gastro esophageal reflux, dysphagia suggest possibility of esophageal ulcer.

Emesis, retching or coughing are associated with Mallory Weiss tear.

History of burns, cerebral vascular disease, head injury, poly trauma suggest possibility of stress ulcer induced UGI Bleeding.

Past History

In case of upper GI bleeding, up to 60% of the patients bleed from the same lesions. [(29)] History of alcohol abuse and liver disorder are associated with varices, portal hypertensive gastropathy. In case of peptic ulcer disease, bleeding is due to H.Pylori infection, NSAID use and smoking.

Co morbid illnesses like coronary artery disease, pulmonary disorder are more susceptible to hypoxemia. Renal and cardiac failure predisposed to volume overload and such patients need invasive monitoring during resuscitation.

Bleeding due to coagulopathies, thrombocytopenia, and hepatic dysfunction are difficult to control such patients need fresh frozen plasma transfusion.

Physical Examination

Physical examination should focus on signs that include the severity of blood loss and helps to localize the source of bleeding.

Physical examination is to assess the general condition of the patient and to assess the signs of shock, hypovolemia which include high pulse rate, low pulse volume,low oxygen saturation, orthostatic hypotension and pallor.

Resting tachycardia indicates mild to moderate hypovolemia.
Orthostatic hypotension indicates up to 15% blood loss.
Supine hypotension indicates about 40% blood loss[30].

If there is abdominal pain which is severe and associated with guarding and rigidity, perforation should be ruled out prior to endoscopy. Signs of chronic liver disease indicates bleeding likely of variceal source which is due to portal hypertension and high mortality is expected.

Palpable epigastric mass, virchow’s node is suspicious of gastric carcinoma.

Purpura, splenomegaly, enlarged lymph nodes denote the possibility of blood dyscrasias.

Stool colour provide clue to the location of bleeding. Black tarry stools denote upper GI bleeding.

Prokinetic Drugs

Both Erythromycin and metoclopramide have been studied in patients with acute upper GI bleeding. The goal is to improve gastric visualization at the time of endoscopy[31]. Erythromycin promotes gastric emptying by acting on motilin receptors.

Single dose of erythromycin 3 mg/kg over 20 to 30 minutes, 20 to 120 minutes before endoscopy improves visibility, shorten endoscopic time and decrease the need for second look endoscopy.

Somatostatin and octreotide

Meta analysis suggest that intravenous administration of somatostatin or its long-acting form octreotide decreases the risk of rebleeding from peptic ulcer. [32]

It is mainly used in the treatment of variceal bleeding and may also benefit in non variceal causes.

Octreotide is given as an intravenous bolus of 20-50 mcg followed by a continuous infusion at a rate of 25 to 50 mcg/hour. [33]

Identification of Bleeding Source

1. Naso gastric Aspiration:

   Aspirate may categorize patients as low/intermediate/high risk groups.

2. Gastric lavage:

   - with saline
   - for better visualization during endoscopy
   - to estimate rapidity of bleeding
   - prevent the development of porto systemic encephalopathy in cirrhosis
   - increases the stomach pH

Endoscopy

Once the patient is hemodynamically stable, endoscopy should be performed to identify the source of bleeding.

Endoscopy can be of early (< 24 hours) and elective (24 – 48 hours). Former is indicated in persistent active bleeding.

Endoscopy offers therapeutic options like glue injections, endo clips application, etc.

Treatment of Non Variceal Bleed

Endoscopic Modalities

a. Injection therapies

- reduce blood flow by local tamponade
- use of vaso constricting agents e.g. epinephrine further reduce blood flow
- 0.5 to 1 ml of 1:20,000 epinephrine by sclerotherapy needle in to four quadrants of ulcer within 1 to 2 mm of bleeding site.
- other agents used are fibrin glue, Human thrombin, sclerosants, absolute alcohol[23]

b. Contact Thermal Therapy: using heater probes

i. Bipolar Cautery

-it can be used perpendicularly or tangentially

-bleeding vessels are compressed and then coagulated

-10-15 watts in duodenum, 15-20 watts in stomach applied over 8-12 seconds until the involved vessels flatten out.

Other techniques:

ii. Laser photo coagulation

iii. Heater probe method

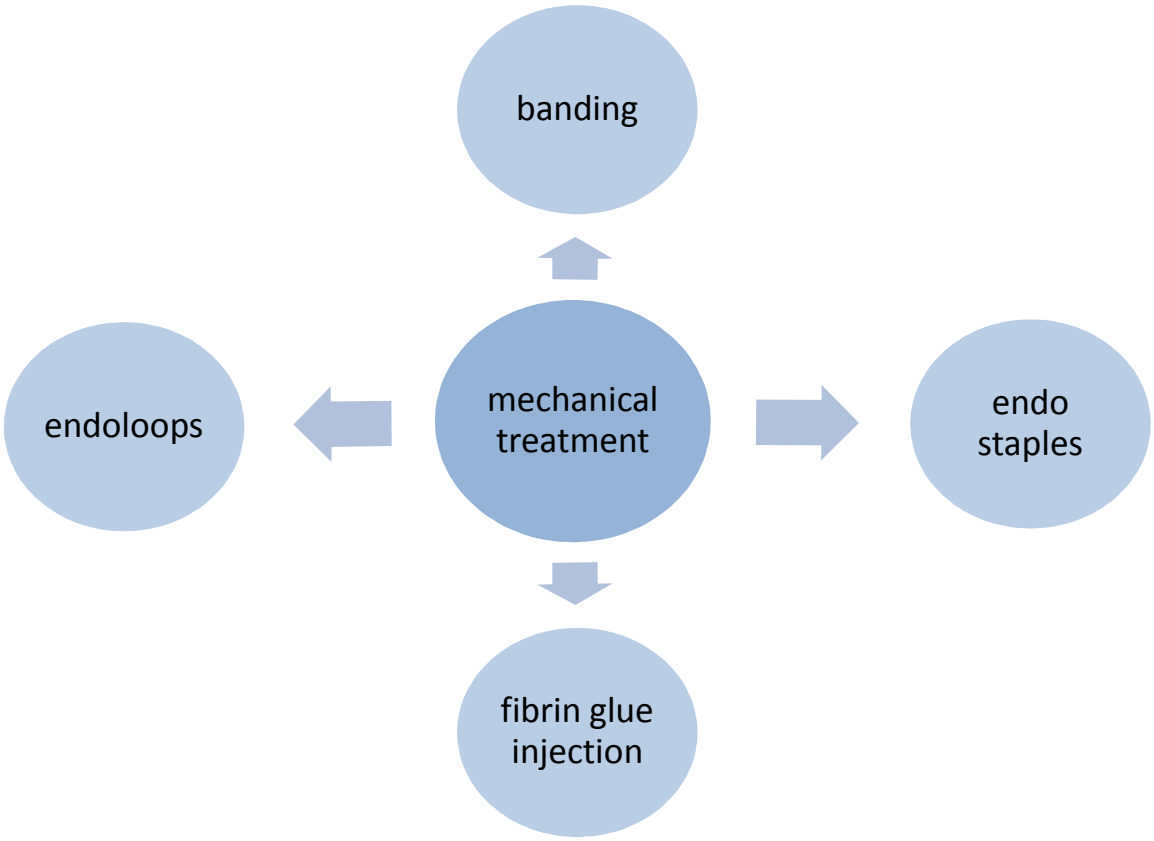
iv. Gold probe

v. Argon plasma coagulation

Mechanical treatment:

- Spraying crystalline collagen, clotting factor or cyanoacrylate tissue glue to bleeding lesion
- Application of hemo clips
- Rebleeding rates are reduced with endo clips.

Other techniques:



Interventional Radiological Procedure

Trans cathetral arterial embolization [34]

Angiography in patients with ulcer more than 2 cm in association with large arteries at the ulcer blood. Also indicated in patients with recurrent bleed despite 2 sessions of endoscopic hemostasis.

Surgical treatment

Emergency surgery

Indications for emergency surgery are:

1. Haemostasis not achieved
2. Rebleeding in elderly, frail patients
3. Rebleeding more than once in a young patient

Elective surgery:

1. When patient has bled from the gastric or duodenal ulcer more than one occasion
2. Visible vessel in the base of the ulcer
3. Bleeding with evidence of gastrinoma
4. Locally confined bleeding associated with malignant ulcerated mass. [23]

Mallory Weiss syndrome/tear [12]

In about 80-90% of the patients, bleeding stops spontaneously. In about 1 to 5% of patients, bleeding recurs. Treatment options include:

i. endoscopic electro-coagulation of the tear

ii. angiography therapy with intro arterial infusion of vasopressin or embolization

iii. operative therapy with over sewing of tear [23]

Erosive gastritis

Erosive Gastritis in sick patients following major surgery, burns, intra cranial disease responds to intravenous H2 receptor antagonist. Sucrafate is also effective. In case of NSAID induced mucosal injury, the following protocols are to be followed. [35]

Clinical Setting	Recommendation
NSAID discontinued	H2 Receptor antogram /PPI
NSAID continued	PPI
Prophylactic therapy	Misoproslot; PPI
H.Pylori infection	H.Pylori regimen

Table 3: Treatment options in erosive gastritis

Pharmalogical Therapy [35]

Drug Type	Examples	Dose
ACID SUPPRESSING DRUGS		
H2 receptor antagonists	Cimetidine	400 mg bid
	Ranitidine	300 mg hs
	Famotidine	40 mg hs
	Nizatidine	300 mg hs
PPIs	Omeprazole	20 mg/d
	Lansoprazole	30 mg/d
	Rabeprazole	20 mg/d
	Pantoprazole	40 mg/d
	Esmoprazole	20 mg/d
MUCOSAL PROTECTIVE AGENTS		
Sucralfate	Sucralfate	1g qid
Prostaglandin analogue	Misoprostal	200µg qid
Bismuth – containing compounds	BSS	

TABLE 4: PHARMACOLOGICAL THERAPY FOR PEPTIC ULCER

Regimens for Eradication of H. Pylori Infection in non ulcer dyspepsia[35]

DRUG		DOSE
Triple Therapy (14 days)		
1. Bismuth Subsalicylate	Plus	2 tab. Qid
- Metronidazole	Plus	250 mg qid
- Tetracycline		500 mg qid
2. Ranitidine Bismuth Citrate	Plus	400 mg bid
- Tetracycline	Plus	500 mg bid
- Clarithromycin or Metronidazole		500 mg bid
3. Omeprazole	Plus	20 mg bid
- Clarithromycin	Plus	250 or 500 mg bid
- Metronidazole	Or	500 mg bid
- Amoxicilline		1 gm bid
QUADRUPLE THERAPY (7-10 DAYS)		
Omeprazole (Lansoprazole)		20 mg (30 mg) od
Bismuth Subsalicylate		2 tab. Qid
Metronidazole		250 mg qid
Tetracycline		500 mg qid

TABLE 5:H. PYLORI REGIMENS

Portal Gastropathy [11]

Treatment options include:

- Beta adrenergic receptor blockers (propanalol)
- Surgical porta caval shunt
- Best treatment option is liver transplantation

Dieulafoy's Lesion [12]

Endoscopic Doppler ultrasound has been used to identify Dieulafoy's Lesion. The site marked with submucosal ink for future retreatment in case of rebleed. Treatment includes injection therapy with thermal probe, clips or band ligation.

Esophagitis [12]

Endoscopic therapy has no role unless a focal ulcer with bleed is visualized. Treatment include PPIs to be taken daily for at least 8-12 weeks. Repeat endoscopy is to exclude underlying Barrett's esophagus.

Upper GI Malignancy[23]

In case of bleeding malignant ulcers, endoscopic haemostasis with monopolar electro cautery, laser, injection therapy, haemoclips can temporarily control active bleeding.

Angiography with embolization should be considered for severe UGIB caused due to malignancy.

External beam radiation can provide palliative haemostasis in case of advanced gastric or duodenal cancer.

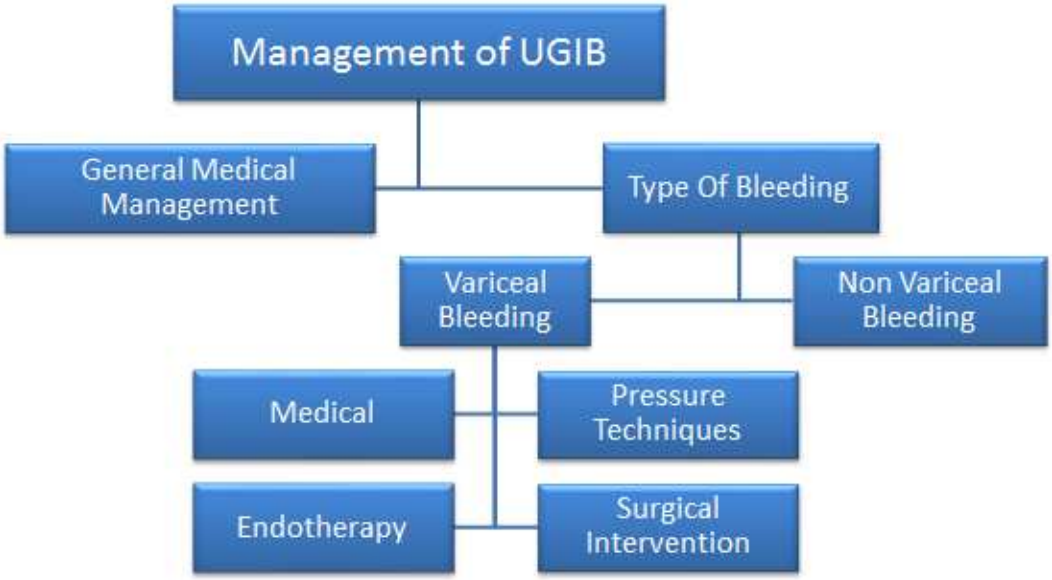
Gastric Antral Vascular Ectasia (GAVE) [12]

Endoscopic therapy with argon plasma coagulation has shown 80% effectivity.

Cameron's Lesions[23]

Long term medical management is usually with iron supplement and oral PPIs alleviate the symptoms.

Variceal Bleed



Medical Management

Vaso constrictors

- Vasopresssin
    - 0.1 to 0.5 units per minute for 4 to 12 hours with short acting nitrates causes suppression of variceal bleed by vasoconstriction of splanchnic vessels whereby decreasing portal venous pressure
- Terlipressin
    - 2 mg bolus followed by 1 mg every 4 to 6 hourly interval for 3 to 5 days
    - It is the only drug that has shown increase in survival[36]

  - Octreotide 50 mcg bolus followed by 50 mcg per hour infusion for 5 days

- Somatostatin
  - 250 mcg bolus followed by 250 mcg hourly infusion decreases the portal pressure and collaterally blood flow by inhibiting release of glucagon[37]
- Non selective beta blockers
  - It has been used extensively in preventing variceal rebleeding [39]
  - Propanalol (40 mg) and nodalol (20 mg) are preferred drugs that are taken once daily
  - Risk of bleeding is greatest at night and hence beta blockers are administered in the evening[40]
  - Patients who do not achieve decrease in the HPVG to less than 12 mm Hg on beta blockers may not respond to endoscopic variceal ligation as well[41]
  - For secondary prophylaxis isosorbide mononitrate may be added to a beta blocker[42]

Pressure techniques

- Sengstaken Blakemore tube
- Minnesota tube
- Linton Nicholas tube

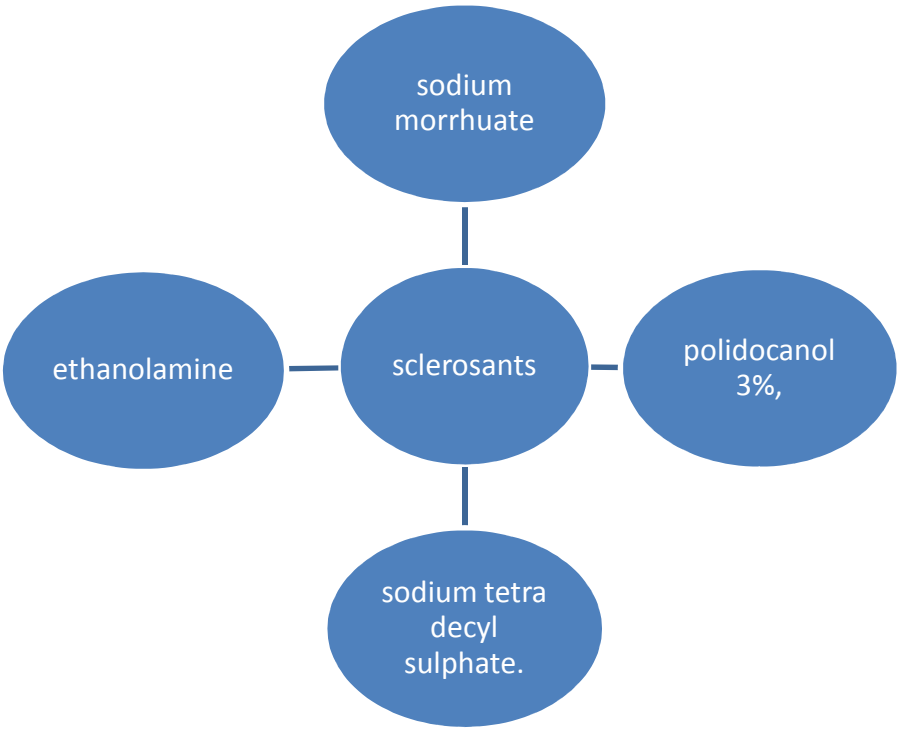
Balloon should be inflated for at least 24 hours and 75% of patients re-bleed after deflation. Balloon tamponade only provides initial control of bleeding in 85 – 98% of cases.

Major complications following balloon tamponade are 30% rate of aspiration pneumonia, esophageal rupture, airway obstruction. Clinical studies do not show significant difference in efficacy between the vasopressin and balloon tamponade. [38]

Endoscopic Sclerotherapy

Sclerosants can achieve haemostasis in 85-95% cases with rebleeding rate of 25-30%.

Complications include esophageal ulcer, perforation, esophageal strictures, mediastinitis, pleural effusion, aspiration pneumonia.

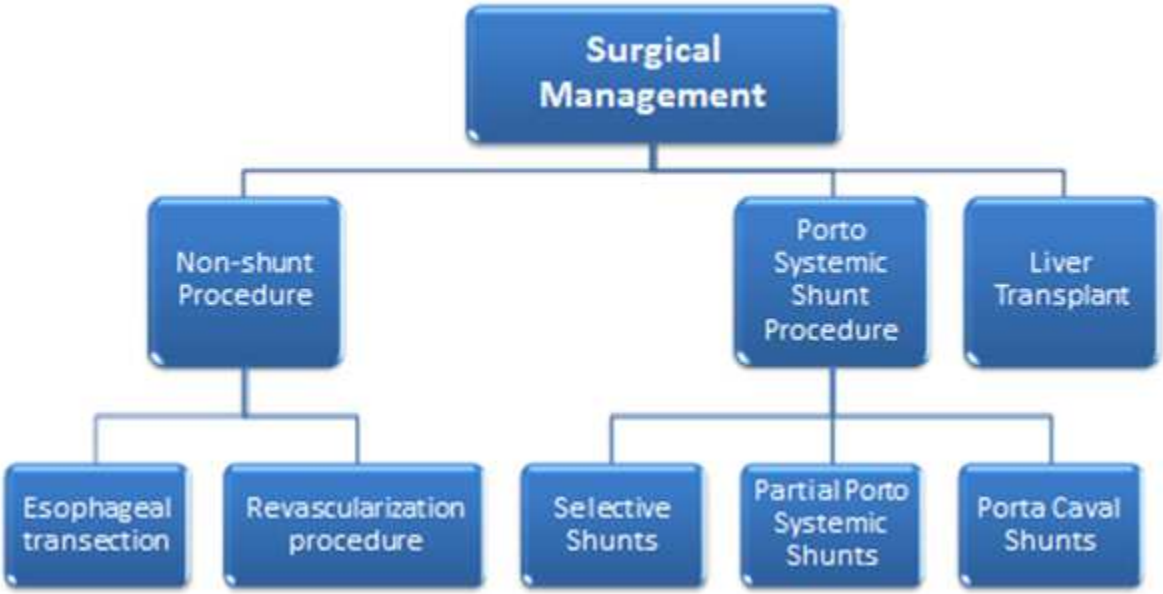


Tissue adhesive glue, N-butryl-2-cyano acrylate preferred in fundal varices.

Banding techniques

Haemostasis achieved in 80-85% of cases with re bleeding rate of 25-35%. Banding techniques are better than sclerotherapy with fewer local complications. Banding of varix subsequently undergoes thrombosis ,sloughing, fibrosis.

Surgical Management



TIPS

Trans Jugular intra hepatic porto-systemic shunt.

Connects intrahepatic branch of portal vein to hepatic vein thus reducing portal vein pressure gradient to less than 12-15 mm Hg.

Complications include bleeding, dye induced renal failure, haemolysis, stent migration.

Used to treat complications of portal hypertension mainly:

i. Refractory variceal bleeding, refractory ascites

ii. Budd chiari syndrome

iii. Hepatic hydro thorax

iv. Hepato renal syndrome

MATERIALS AND METHODS

In this prospective study, cases admitted with upper GI bleed in various surgical units, among 50 patients selected randomly, in Government Mohan Kumaramangalam Medical College Hospital. The cases were evaluated through:

1. Proper history taking
2. Thorough clinical examination
3. Endoscopic examination
4. Other relevant investigations
5. Outcome at the end of the hospital stay

For the purpose of analysis, the patients with acute upper GI bleeding were divided into 7 groups.

1. Duodenal ulcer
2. Gastric ulcer
3. Esophageal varices
4. Gastric erosion
5. Carcinoma stomach
6. Miscellanous cause
7. Source Unknown

Miscellaneous group included

1.esophagitis,

2.esophageal carcinoma,

3.telangiectasia,

4.Mallory Weiss tear.

Patients from both genders were admitted in our hospital with history of hematemesis or malena who are hemodynamically stable after resuscitation were included in this study.

Exclusion criteria were:

1. Patients less than 15 years
2. Patients who were not stabilized within 48 hours of resuscitation
3. Patients who have contra indications to endoscopy
4. Patients previously diagnosed to have GI malignancies under treatment
5. Upper GI bleed due to corrosive poisoning
6. Upper GI bleed due to medical cause [bleeding disorder ,drug induced]

A printed proforma, enclosed in Appendix 1, was made to record all information collected from the patient and for analysis.

OBSERVATION AND RESULTS

Etiological Pattern of upper G I bleed:

S.No.	Etiology	No. of Patients	Percentage
1	Duodenal Ulcer	17	34
2	Gastric Ulcer	8	16
3	Esophageal varices	10	20
4	Gastric Erosion	5	10
5	Gastric Carcinoma	2	4
6	Miscellaneous	5	10
7	Unknown	3	6

Table 6: Etiological Pattern of Upper G I Bleed

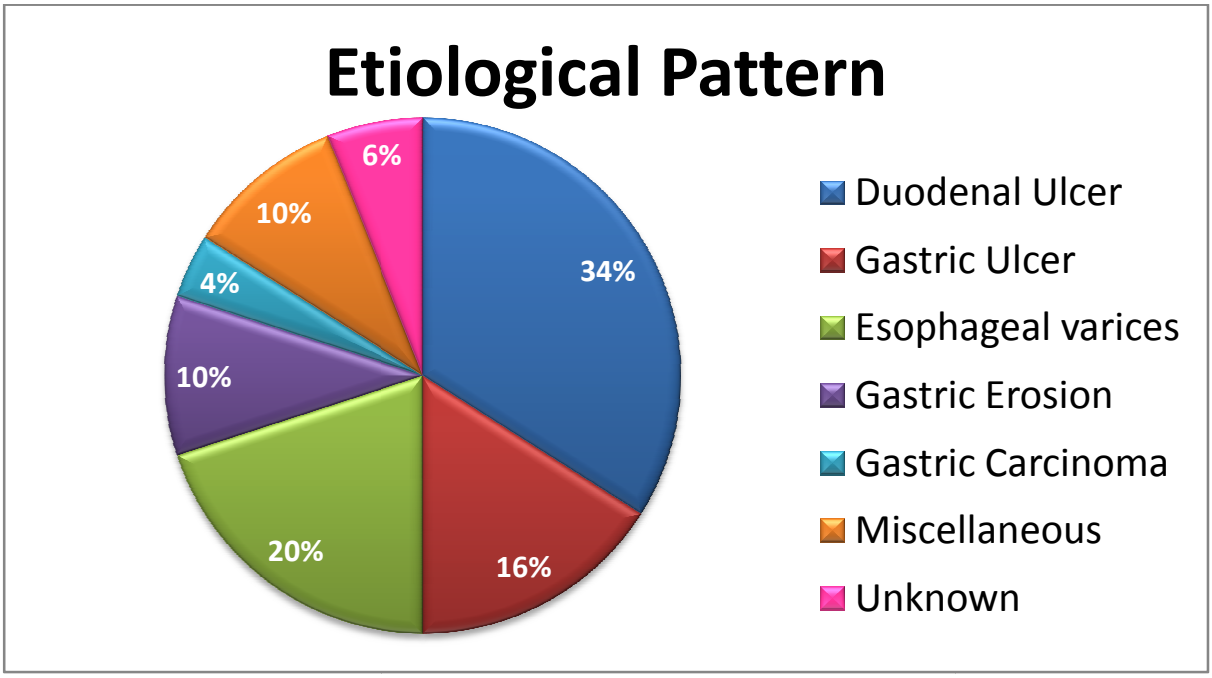


Fig 3: Etiological Pattern of Upper G I Bleed

The final diagnosis was made after upper G I endoscopy. Among the various etiology, peptic ulcer was the commonest cause (50%). Duodenal ulcer contributed34% followed by variceal bleeding 20%.

Upper GI Bleed in Relation to Age

Age Group	No of Cases	Percentage
15-24	4	8
25-34	4	8
35-44	10	20
45-54	13	26
55-64	9	18
65-74	9	18
75-84	1	2

Table 7: Upper GI Bleed in Relation to Age

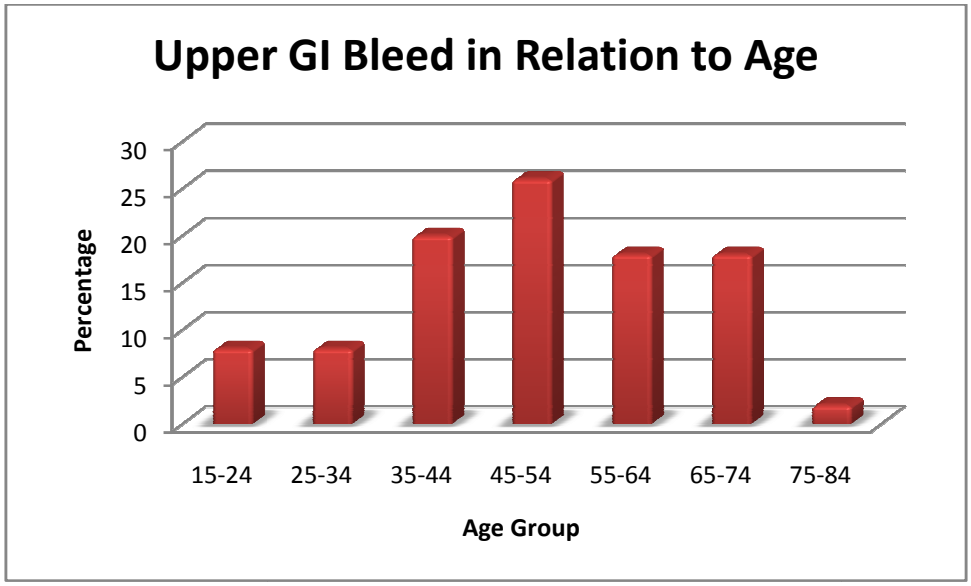


Fig.4: Upper GI Bleed in Relation to Age

Over all the commonest age group who suffered from upper GI bleed were 45-54 year group (26%).Bleeding due to peptic ulcer was common in younger age group,malignancy in elderly age group.

Upper GI Bleed in Relation to Gender

Etiology	Gender	
	Male	Female
Duodenal Ulcer	15	2
Gastric Ulcer	6	2
Esophageal varices	8	2
Gastric Erosion	4	1
Gastric Carcinoma	1	1
Miscellaneous	3	2
Unknown	3	0

Table.8: Upper GI Bleed in Relation to Gender

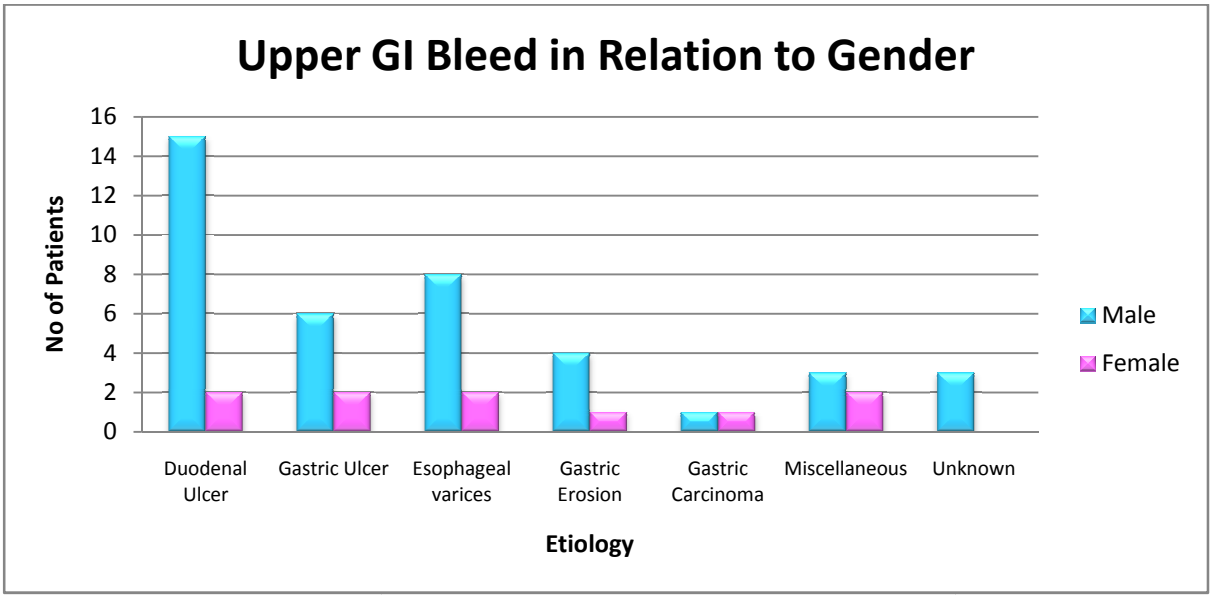


Fig 5: Upper GI Bleed in Relation to Gender

Upper GI bleed was common in male (80%). Female contributed 20%.

Male: Female ratio was 4:1.

Habitat

Etiology	Urban	Rural
Duodenal Ulcer	6	11
Gastric Ulcer	3	5
Esophageal varices	4	6
Gastric Erosion	1	4
Gastric Carcinoma	1	1
Miscellaneous	2	3
Unknown	1	2

Table 9: Upper GI Bleed in Relation to habitat

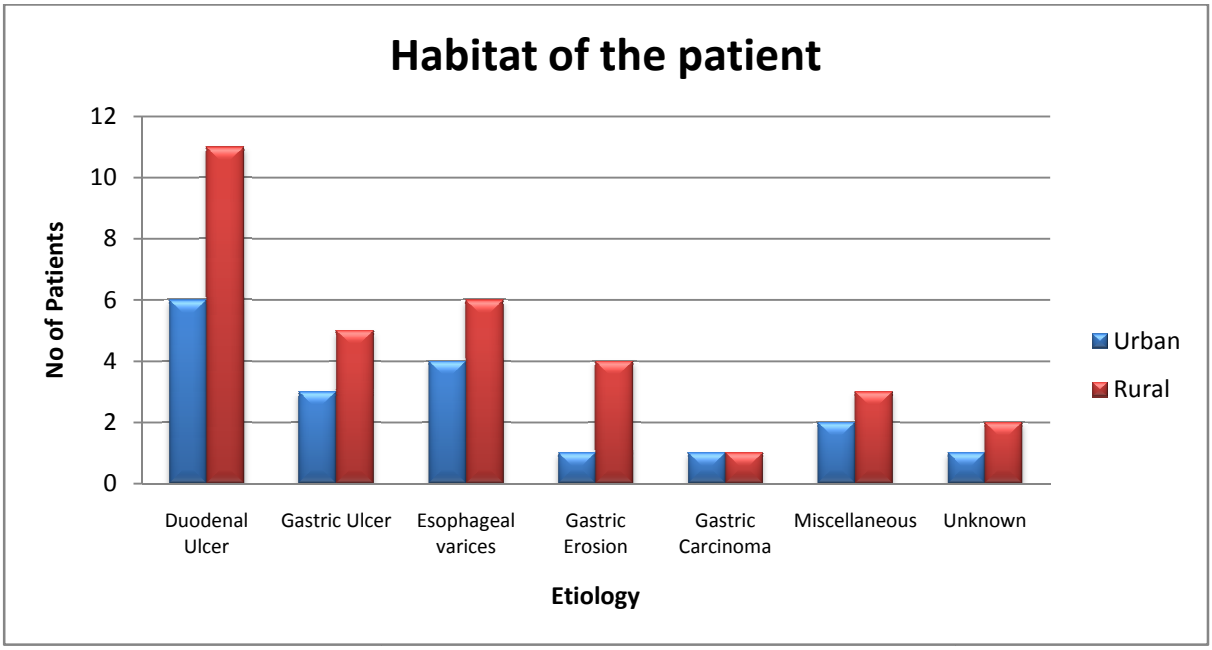


Fig 6: Upper GI Bleed in Relation to habitat

Rural population (64%) suffered upper GI bleed more common than the urban population (36%).

Etiological Agents

Etiology	Smoking	NSAIDS	Steroids	Alcohol
Duodenal Ulcer	11	8	3	4
Gastric Ulcer	4	3	1	1
Esophageal varices	4	0	1	6
Gastric Erosion	1	4	1	2
Gastric Carcinoma	1	0	0	0
Miscellaneous	2	1	0	1
Unknown	1	3	0	0

Table 10: Upper GI Bleed in Relation to etiological agents

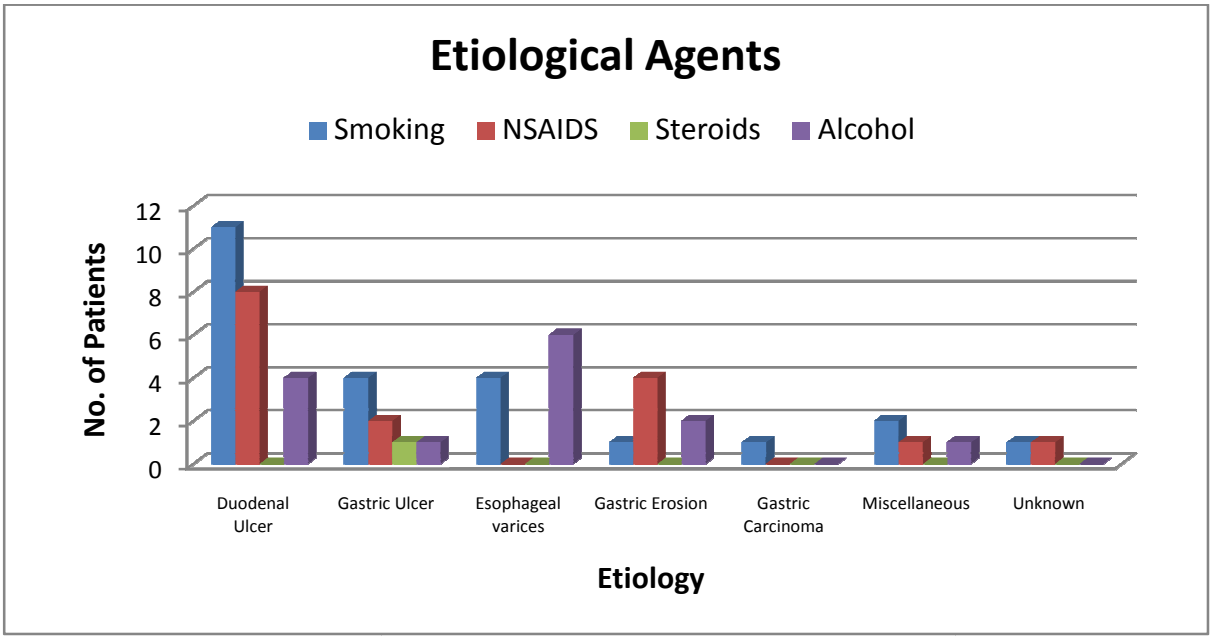


Fig 7: Upper GI Bleed in Relation to etiological agents

Correlation of upper GI bleed with smoking was found 11(65%), 4(50%), 1(25%) cases of duodenal ulcer, gastric ulcer, erosive gastritis respectively. NSAIDs were found 8(47%), 3(38%), 4(80%) of duodenal ulcer, gastric ulcer, erosive gastritis respectively.

Clinical Presentation of the Patients

Symptoms	Hematemesis		Malena		Hematemesis & Malena	
	No of Patients	Percentage	No of Patients	Percentage	No of Patients	Percentage
Duodenal Ulcer	4	24	3	17	10	59
Gastric Ulcer	3	38	1	13	4	50
Esophageal varices	4	40	2	20	4	40
Gastric Erosion	1	20	1	20	3	60
Gastric Carcinoma	0	0	1	15	1	50
Miscellaneous	2	40	1	20	2	40
Unknown	1	33	0	0	2	67

Table 11: Clinical Presentation of the Patients

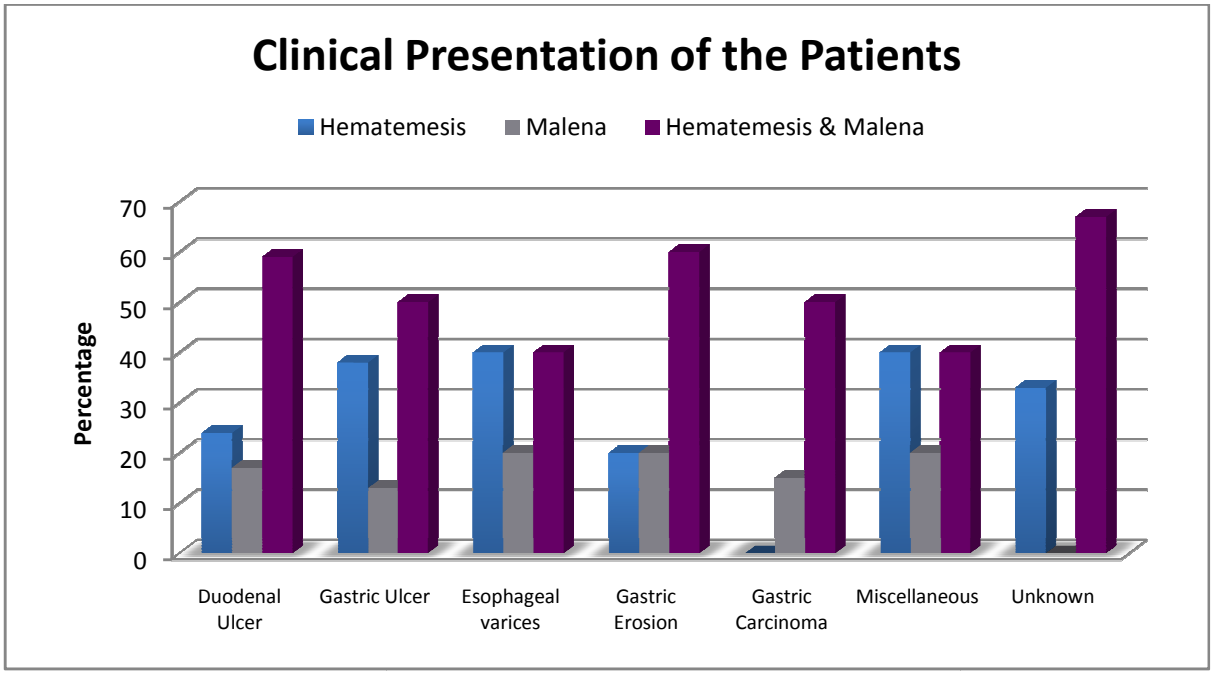


Fig.8: Clinical Presentation of the Patients

Hematemesis ,malena and both hematemesis and malena were the presenting features of 30%, 18% and 52% of the patients.

Among them 4 (24%) patients of DU, 3 (38%) patients of GU & 4 (40%) patients of oesophageal varices presented with hematemesis and 3 (17%) patients of DU 1 (13%) patients of GU & 2 (20%) patients of oesophageal varices presented with malena 10 (59%) patients of DU presented with hematemesis and malena.

Past History

Etiology	Epigastric Pain		Hematemesis/Malena		Jaundice		Loss of weight	
	No of Patients	Percentage	No of Patients	Percentage	No of Patients	Percentage	No of Patients	Percentage
Duodenal Ulcer	15	88	2	12	0	0	2	12
Gastric Ulcer	6	75	0	0	0	0	6	75
Esophageal varices	2	20	3	30	8	80	5	50
Gastric Erosion	4	80	0	0	0	0	0	0
Gastric Carcinoma	0	0	0	0	1	50	2	100
Miscellaneous	1	20	0	0	0	0	0	0
Unknown	1	33	0	0	0	0	0	0

Table .12: Upper GI Bleed in Relation to past history

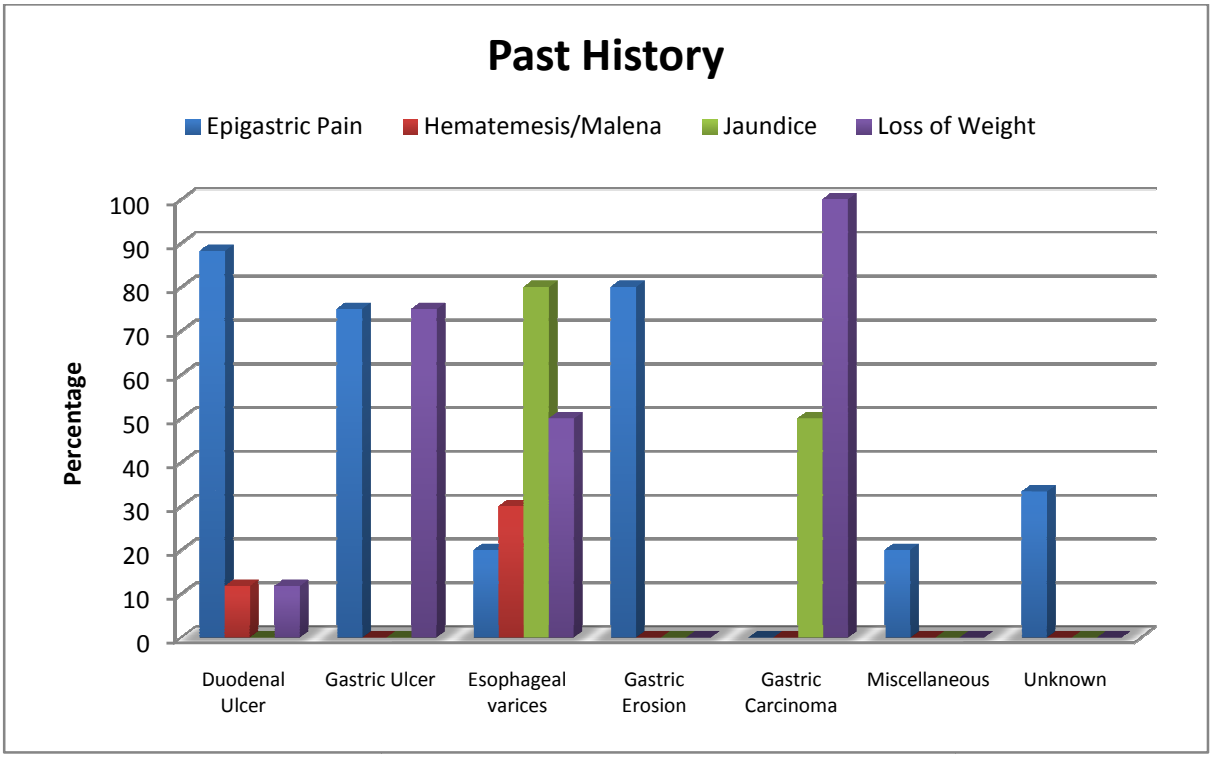


Fig 9: Upper GI Bleed in Relation to past history

Past history of epigastria pain was noted 15 (88%) and 6 (75%) cases of duodenal ulcer and gastric ulcer patients respectively. Past history of jaundice was obtained in 8 (80%) cases with oesophageal varices. Past history of hematemesis and malena was obtained in 2 (12%) & 3 (30%) cases of duodenal ulcer and oesophagcal varices.

Correlation with Clinical Signs

Signs	No of Patients	Percentage
Anemia	37	74
Epigastric Tenderness	30	60
Splenomegaly	9	18
Ascites	10	20
Hepatic Failure	9	18

Table.13: Upper GI Bleed in Relation to clinical signs

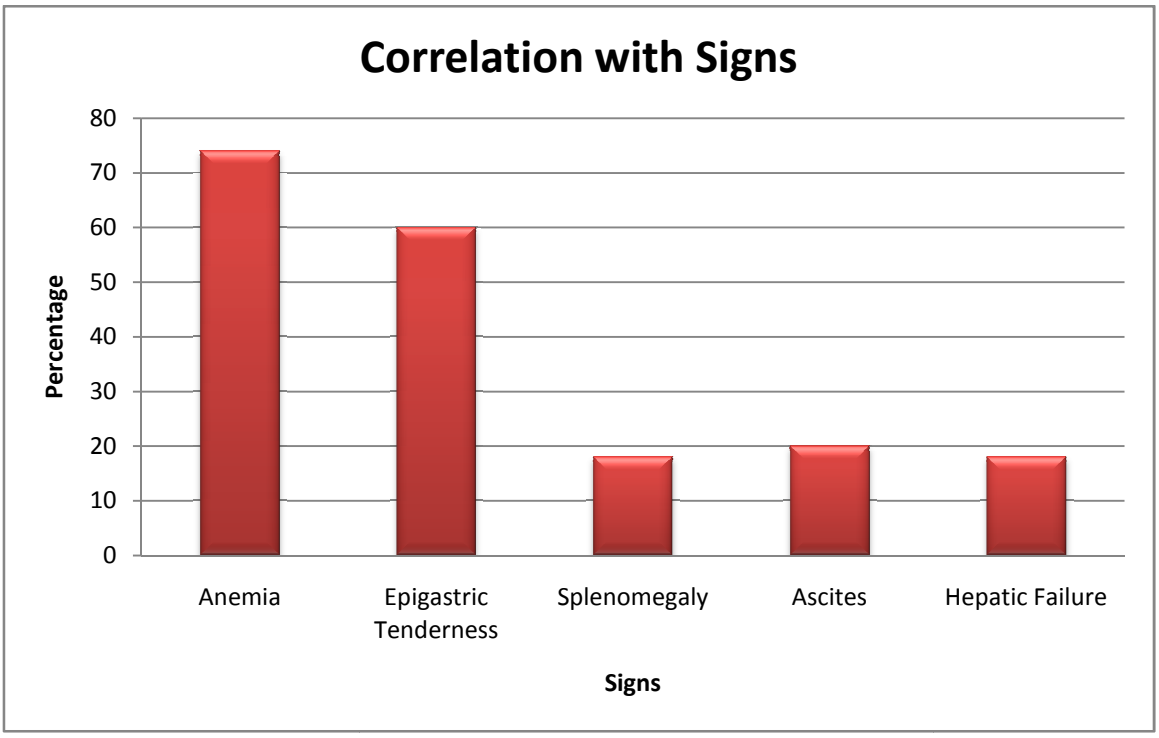


Fig 10: Upper GI Bleed in Relation to clinical signs

Anemia, epigastric tenderness, splenomegaly were found in 74%, 60% and 18% cases respectively.

Relation with Blood Group

Etiology	Blood Groups							
	O		A		B		AB	
	No of Patients	Percentage	No of Patients	Percentage	No of Patients	Percentage	No of Patients	Percentage
Duodenal Ulcer	8	47	1	6	4	24	4	24
Gastric Ulcer	3	38	1	13	2	25	2	25
Esophageal varices	5	50	1	10	3	30	1	10
Gastric Erosion	2	40	1	20	2	40	0	0
Gastric Carcinoma	0	0	1	50	0	0	1	50
Miscellaneous	2	40	0	0	2	40	1	20
Unknown	2	67	0	0	1	33	0	0

Table 14: Upper GI Bleed in Relation to blood group

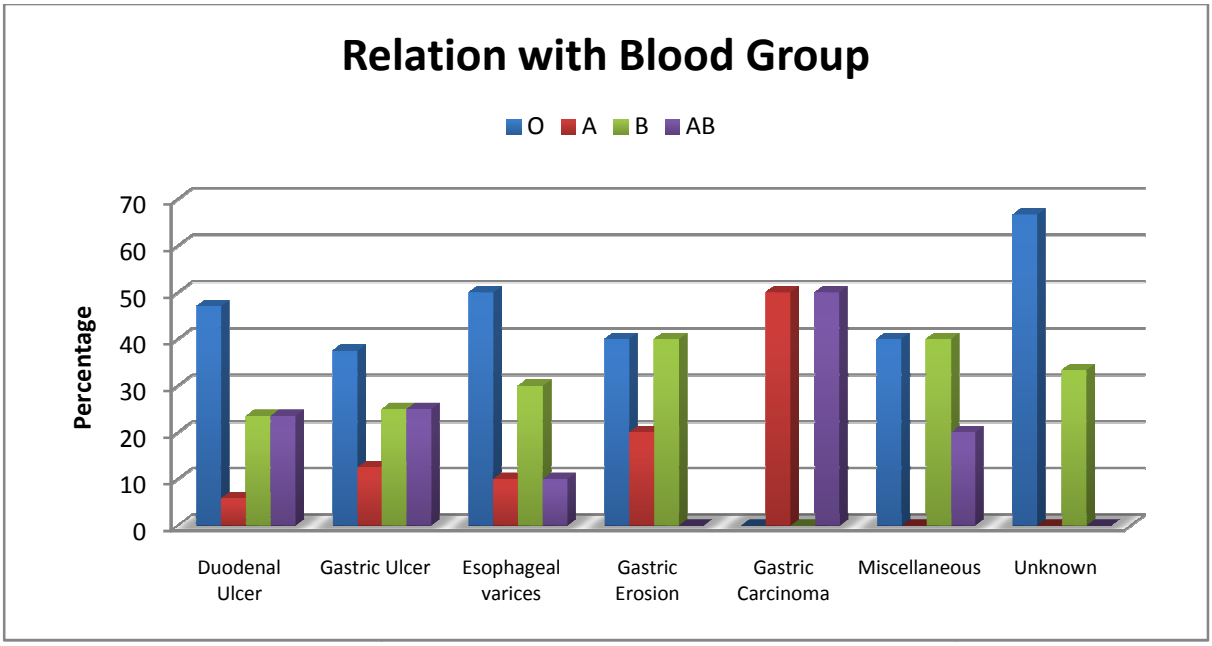


Fig 11: Upper GI Bleed in Relation to blood group

As a whole, patients with “O” blood group suffered more 22(44%) from hematemesis & malena with especially due to duodenal ulcer (47%).

Naso Gastric Aspirate

NGA	No of Patients	Percentage
Clean	7	14
Coffee Ground	16	32
Red Blood	27	54

Table.15: Nature of Naso Gastric Aspirate

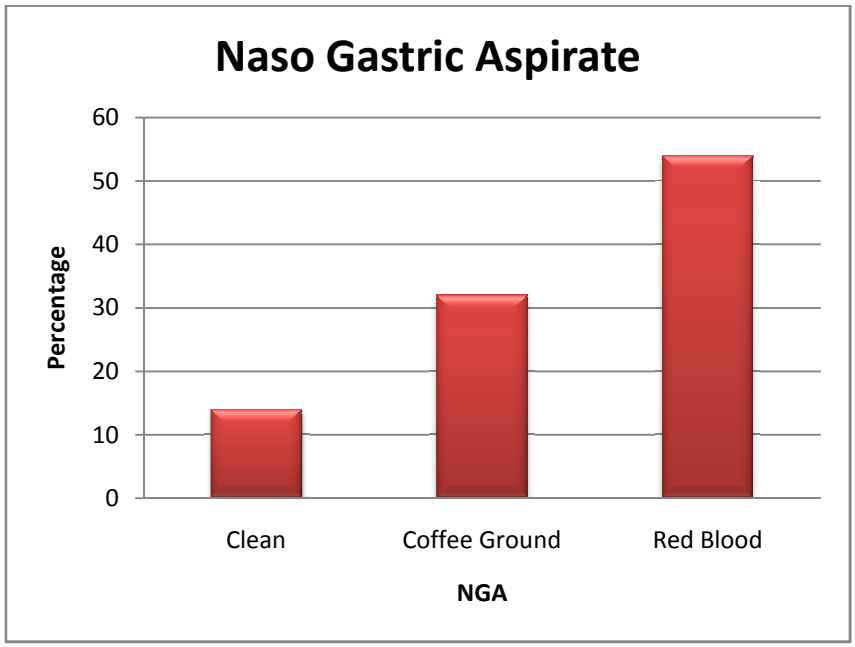


Fig 12: Nature of Naso Gastric Aspirate

Nasogastric aspiration shows 86% of the patients having fresh or coffee ground colour of aspirated material which is suggestive for hematemesis.

Rockall Score Interpretation

Score	No. of Patients	Percentage (%)
Low Risk (< 3)	10	20
Moderate Risk (3 – 5)	27	54
High Risk	13	26

Table.16: Rockall Score Interpretation

Rockall Scoring and its adverse outcomes

Variable	Low Risk	Medium Risk	High Risk
Score	0 - 2	3 - 5	6 – 11
No. of Patients	10	27	13
Blood Transfusion	4	16	13
Re admission	-	3	3
Repeat Endoscopy	1	3	2
Re bleed	1	7	3
Death	-	-	2

Table.17: Rockall Scoring and its adverse outcomes

Rock all score was calculated and interpreted. Majority of patients 27(54%) were in the medium risk category .of which 4(40%), 16(59%), 13(100%) of patients belonging to low risk, medium risk, high risk groups required blood transfusion.1(10%),7(26%),3(23%) of low risk, medium risk, high risk groups had rebleeding.

Various Management Modalities

Type of Therapy	Total	Percentage
Proton Pump Inhibitors	37	74
Vasopressin	2	4
Octreotide	12	24
Sclerotherapy	10	20
Banding	9	18
Surgery	2	4

Table.18: Various Management Modalities

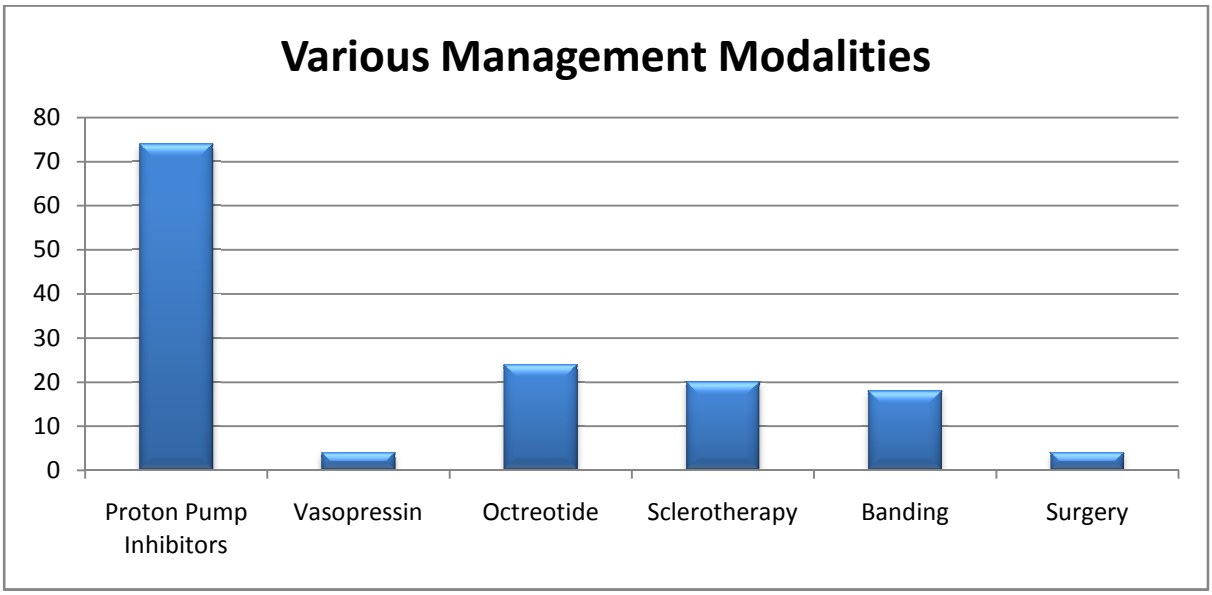


Fig.13: Various Management Modalities

In managing the cases with hematemesis and malena 65 patients (65%) were treated with 37 patients (74%) were treated with proton pump inhibitor (e.g. pantoprazole). Among the clinically suspected oesophageal varices patients two were treated with vasopressin and twelve were treated with octreotide, 9 were treated with banding. Among the endoscopically diagnosed ulcer bleeders 10 were treated with sclerotherapy.

Recurrent Bleed

Etiology	Conservative	Sclerotherapy	Banding	Total	Percentage
Duodenal Ulcer	1	0	0	1	5.9%
Gastric Ulcer	1	0	0	1	2.5%
Varices	1	1	1	3	30%

Table.19: Recurrent Bleed

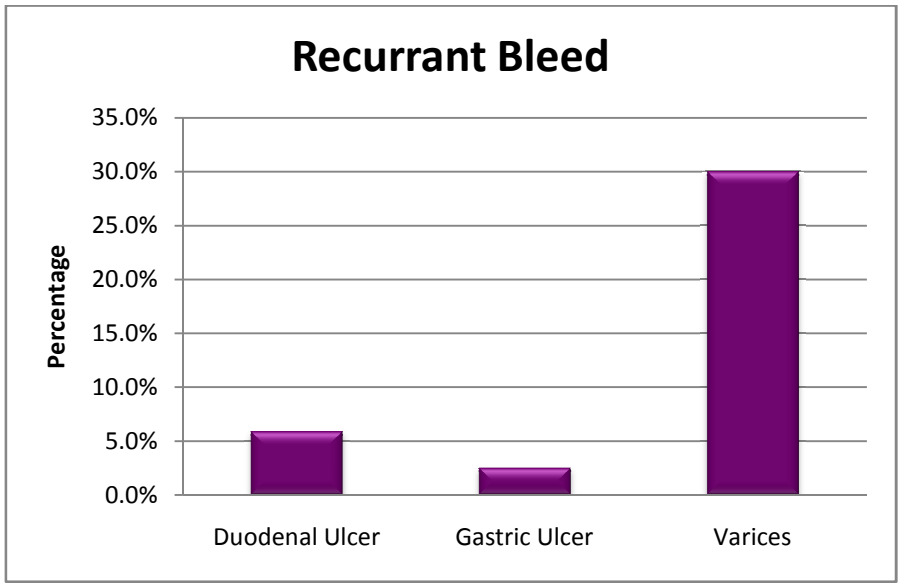


Fig.14: Recurrent Bleed

During the hospital stay, 1(5.9%), 1(2.5%), 3(30%) of duodenal ulcer, gastric ulcer and esophageal varices developed recurrent bleed.

Hospital Stay

Etiology	No of Cases	Mean Stay (days)
Duodenal Ulcer	17	5
Gastric Ulcer	8	4
Esophageal varices	10	9
Gastric Erosion	5	3
Gastric Carcinoma	2	9
Miscellaneous	5	5
Unknown	3	2

Table.20: Mean hospital stay

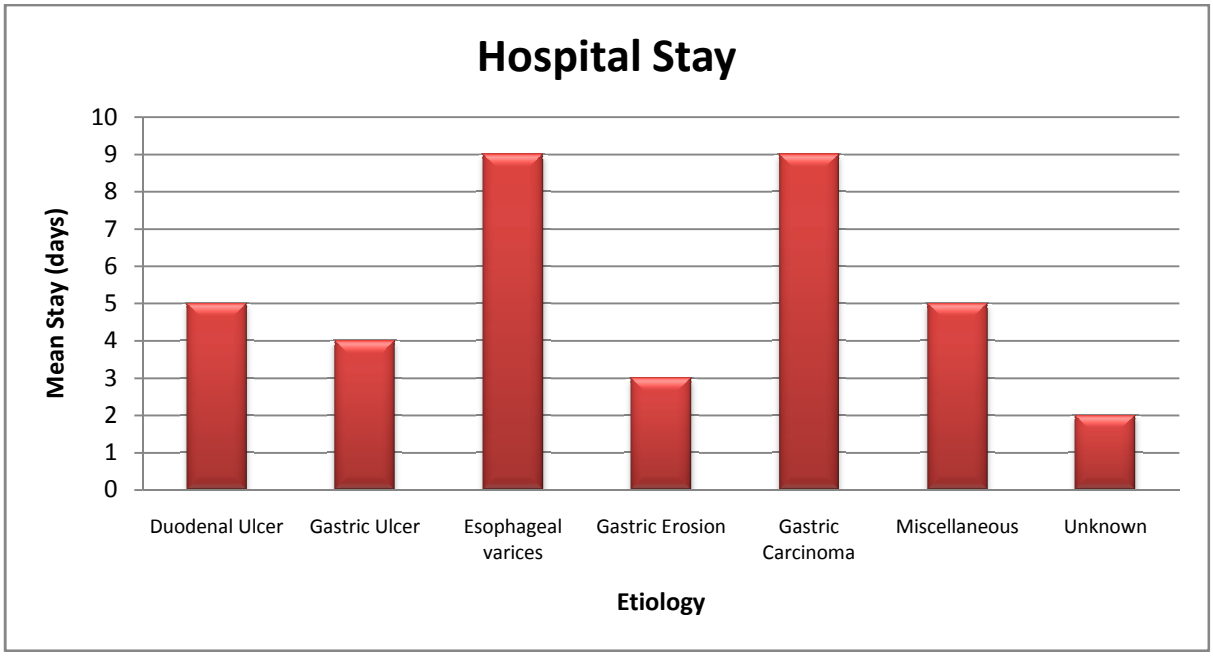


Fig 15: Mean hospital stay

Mean hospital stay of duodenal ulcer was 5 days, gastric ulcer was 4 days and for esophageal varices was 9 days.

Outcome

Etiology	Recovered	Expired	Mortality
Duodenal Ulcer	16	1	5.9%
Varices	10	1	10%

Table.21:Patient outcome

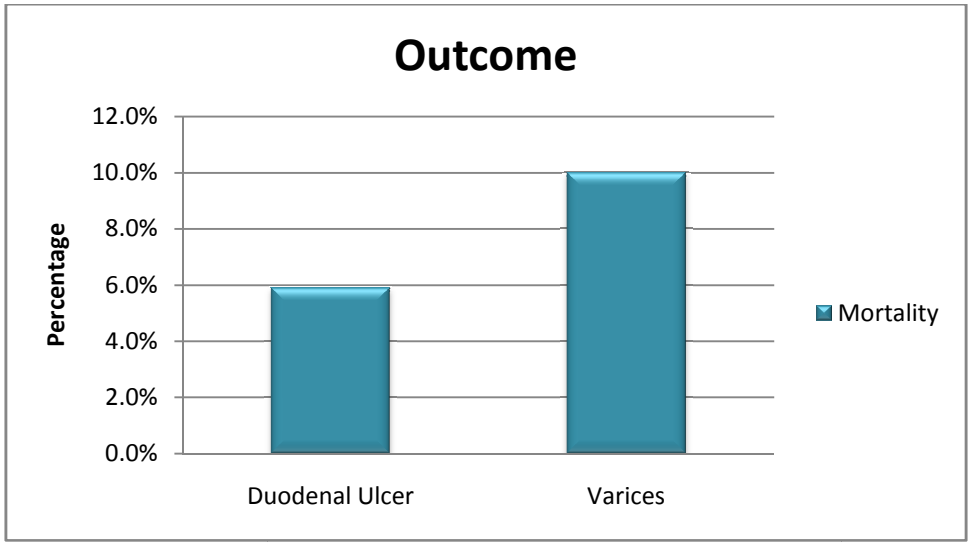


Fig 16: Patient outcome

Overall mortality due to Upper GI Bleed is 4% of which duodenal ulcer had 1(5.9%), esophageal varices had1 (10%) mortality rate.

DISCUSSION

In this study, the mean age of the patients presented with upper GI bleeding was 44.56 and was 49.82 years for duodenal ulcer and 52.25 years for gastric ulcers(.table 6). Mamun[46] in his study showed that the mean age of the patients with upper gastrointestinal haemorrhage was 35.65 years and 49.40 years for duodenal ulcer and gastric ulcer respectively. Islam[19] found that the mean age incidence of duodenal ulcer was 38.4 years and gastric ulcer 47.0 years. Our findings in the current study were very similar to the above studies.

In the current study male female ratio is 4 for bleeding peptic ulcer. This ratio was 7.5 & 3 for duodenal ulcer and gastric ulcer respectively (Table-7). These findings are similar to those of Mamun[46].Alam[47] and Miah[48] found male to female ratio of hematemesis and malena 6.6, 7.5 and 7.3 respectively . Zimmerman et.al [49] also found male preponderance among the bleeders. This increased incidence of the ratio in our country reflects that males are more sufferers.

The ratio of bleeding duodenal ulcer to gastric ulcer was 2.13(Table-6) in the present study. Alam[47]Mamun[46] and Miah[48] found the ratio 5.7, 11.66 and 3.5 respectively. Ratio of bleeding between DU to GU in the western countries varies from 0.8 to 1.2 only. The reason for the higher proportion of the duodenal ulcer to gastric ulcer bleeding in our country than that of the western countries

may be due to geographical and racial variation as well as poor health care facilities. [50]

Peptic ulcer is the commonest cause of hematemesis and malena. [51] The majority of such ulcers are found in the duodenum. Peptic ulcer disease is responsible for approximately half of all hospital admission with upper gastrointestinal haemorrhage in the western countries. In the present study peptic ulcer disease is the commonest cause of hematemesis & malena and comprises 50% and duodenal ulcer contributes to 34% of all cases and gastric ulcer is 16%. Saowaros at all in a study of endoscopic review of 5000 patients with upper GI haemorrhage in Thailand it was found 51.24% of causes were due to peptic ulcer disease. Zimmermair at al, [49] in Jerusalem, in a study found 39.5% duodenal ulcer and 16.9% gastric ulcer. Alam [47] in his study showed PUD responsible for 50% for hematemesis & 58% was the leading causes of upper GI haemorrhage which is similar to the study of Mamun[46] and Alam[47]

Variceal bleeding is an important cause of upper GI haemorrhage. In our present study 20% cases (Table-6) were due to variceal bleeding. It was similar to the findings of Alam[47], Miah[48] ,Mamun[46] and Zimmennun in Jerusalem[49], In USA it is similar to our study (20%).Erosive gastritis is an important cause of upper GI haemorrhage. It is implicated in up to 20% of cases of acute upper GI bleeding with an over all mortality of 10%. Mamun[46], in his

study, found 16% cases of upper GI hemorrhage was due to gastric erosion which was similar to those of Alam[47] & Zimmerman et al[49]; whereas Miah [48] in his study found only 10%..in our study it was about 10% similar to that of Miah[48] and Alam[47] but dissimilar to those of Mamun[46] and Alam[47]. This may be due to small number of study population in this study. If the study would done in a large scale this picture could have been changed. Again may be due to intake, of newer NSAIDs and are less irritant to the gastric mucosa and increase consciousness need among both urban and ruler people about adverse effect of NSAIDs. The source of bleeding could not be detected at endoscopy in 3 (6%) cases. Mamun[46]Miah[48] and Alam[47] were unable to detect the source of bleeding in: 3(4%) 4(8%) and 3(6%) cases respectively.

In the miscellaneous group of this study GIT malignancy contributes 5 (10%) of Cases with hematemesis and malena which is similar to the study of Miah[48] and Alam[49]. In this group we had 2(4%) case of oesophagitis, 1(2%) cases of Mallory weis tear,1(2%) esophageal carcinoma,2(4%) gastric carcinoma1(2%) telangiectasia, which is similar to that of Miah[48] & Alam[47] Correlation of upper GI bleed with smoking was found 11(65%),4(50%),1(25%) cases of duodenal ulcer,gastric ulcer, erosive gastritis respectively., Mamun[46],alam [47] and Miah[48] found 40%, 45.83% and 76% cases with duodenal ulcer respectively and 66.6%, 40% and 80% cases with gastric ulcer respectively. Our findings

were similar to those of Alam[47] and Mamun[46]. Islam, [19] found 50% and 50% of patients associated with smoking for DU and GU respectively.

Intake of ulcerogenic drugs especially aspirin and other NSAIDs assumed to cause peptic ulceration in 15-25% of patients [52] and were associated with relative risk for ulcer bleeding of up to 5 fold. Chalmers et.a'l,[53] in their met analysis recorded that the incidence of upper Gl bleeding was three times more common in patients taking NSAIDs, than in the control group. Up to 22% of bleeding episodes may be due to these drugs with a mortality of 10%. [(54)] NSAIDs were found 8(47%), 3(38%), 4(80%) of duodenal ulcer, gastric ulcer, erosive gastritis respectively.

Miah [(48)] in his study showed 25% of gastric ulcer and 75% of cases of erosive gastritis with upper GI haemorrhage had history of taking NSAIDs. Alam[(47)] in his study has shown that upper GI bleeding due to erosive gastritis occurred after exposure to aspirin and other NSAIDs were 60% of the patients. These findings are similar to our finding regarding with erosive gastritis. In our series we found 6(12%) history of taking steroid in patients with upper GI haemorrhage. In these study group rural population (64%) suffered upper GI bleed more common than the urban population(36%).which is similar to Alam [47] which was about 58%.

In respect of ABO blood group system, As a whole patient with "O" blood group suffered more 22(44%) from hematemesis & malena with especially due to duodenal ulcer (47%). This findings is similar to those of Mamun[46], Alam[47] Miah[48] and Alam[47] who found 39.1% 57.1% 61.5% and 60% respectively of patients with DU bleeders had blood group "O".

In this study, hematemesis, malena and both hematemesis and malena were the presenting features of 30%, 18% and 52% of the patients. Among them 4(24%) patients of DU, 3 (38%) patients of GU & 4 (40%) patients of oesophageal varices presented with hematemesis and 3 (17%) patients of DU 1 (13%) patients of GU & 2 (20%) patients of oesophageal varices presented with malena 10 (59%) patients of DU presented with hematemesis and malena.(Table.10)

Miah [48] in his study found 15.3% of DU, 25% of gastric ulcer and 37.0% of oesophageal varices group had history of hematemesis and malena. Alam[47] in his study found 15% of DU and 37.5% of variceal bleeding. our findings regarding duodenal ulcer and variceal bleeders are similar to that Miah[48] and Alam[47].

Miah[48] showed DU patients presented with both hematemesis and malena in 46.3% cases, malena in 38.4%, and hematemesis in 15% cases. Alam[47] showed

that DU patients presented with hematemesis in 17.1 %cases, malena in 31.4% cases and both haemeaemesis and malena in 51.1% cases. Bleeding gastric ulcer & oesophageal varices presented equally 33.3% with both hematemesis and malena. Variation in presentation in cases of upper GI haemorrhage different studies maybe explained by the fact that haemetemesis and malena is dependent upon the rate, amount and site of bleeding,

Anemia, epigastric tenderness, splenomegaly were found in 74%, 60% and 18% cases respectively

Epigastric tenderness was present in 60% of patients of this series; it is a significant physical finding in active peptic ulcer disease. Miah[48] found epigastric tenderness in 70% of cases. Alam[47] found epigastric tenderness in 76.6% of cases. Our finding is similar to that of Miah and Alam.

Splenomegaly was present in all cases (80%) of variceal bleeding. Stigmata of chronic liver disease with splenomegaly helps in diagnosing a case of cirrhosis clinically. Splenomegaly is the most consistent finding of the patients presented with variceal bleeding. Splenomegaly is also a feature of haematological malignancy.

Nasogastric aspiration shows 86% of the patients having fresh or coffee ground colour of aspirated material which is suggestive for hematemesis. (Table 14) which is similar to the findings of other studies [47,48]. Haemoglobin percentage does not change immediately after hematemesis and malena. It usually takes 48 hours for haemodilution to occur. In our series the mean Hb% was 7.50gm/dl.

Rock all score was calculated and interpreted majority of patients 27(54%)were in the medium risk category .of which 4(40%), 16(59%), 13(100%) of patients belonging to low risk, medium risk ,high risk groups required blood transfusion.1(10%), 7(26%), 3(23%) of low risk, medium risk, high risk groups had rebleeding.

During the hospital stay, 1(5.9%), 1(2.5%), 3(30%) of duodenal ulcer, gastric ulcer and esophageal varices developed recurrent bleed.

Kaniz [54] in a retrospective study of 126 to 1996 in Holy Family Red Crescent Hospital, Dhaka, found recurrent bleeding in 19.04% cases. Our finding has similarity with that of Kaniz[54]. Study in UK is also has similarity to our study[66].

Hematemesis and malena result from various pathological conditions such as peptic ulcer, erosive gastrodudenitis, oesophagitis, oesophgeal varices, ca-stomach ca-oesophgus & blood dscrasias. A single therapeutic modality should not consistently reduce bleeding from such an array on selected subgroup of patients such as portal hypertensive naturopathy. Drug treatment may

significantly reduce mortality and morbidity. In our present series we used H2 receptor antagonist routinely in all variceal bleeding 10 patients although there is no controlled evidence of benefit of use of H2 receptor antagonist or antacid in variceal bleeding though they are used to prevent bleeding from stress induced acute mucosal ulcer.

In managing the cases with hematemesis and malaena 65 patients (65%) were treated with 37 patients (74%) were treated with proton pump inhibitor (e.g. pantoprazole). Among the clinically suspected oesophageal varices patients two were treated with vasopressin and twelve were treated with octreotide, 9 were treated with banding. Among the endoscopically diagnosed ulcer bleeders 10 were treated with sclerotherapy.

The H2 receptor antagonist drugs block the production of acid by the parietal cells and heal 60-90% of duodenal ulcer over 4to 8 weeks with compared with 35-45% with placebo plus antacid as required for relief of pain[55]

Patients with hematemesis and malena need blood transfusion for resuscitation. Patients need blood transfusion until their vital signs are stable, bleeding ceases and enough red cells in circulation to provide adequate oxygenation[54] .In the present study highest blood transfusion was needed in the variceal group(4.50

unit) followed by duodenal ulcer (3.5 unit). This finding is similar to theta of Miah[48], Alam[47].

Mean hospital stay of duodenal ulcer was 5 days, gastric ulcer was 4 days and for esophageal varices was 9 days.

Overall Mortality due to Upper GI Bleed is 4% of which duodenal ulcer had 1(5.9%) , esophageal varices had1(10%) mortality rate. Miah[48] found that the mortality was only 6%.

The high rate of mortality from variceal bleeding may be due is the advanced stage of cirrhosis of liver and development of complications such as hepatic encephalopathy, renal failure, rebleeding and hepatocellular carcinoma etc. The mortality from DU diseases is due to having co-morbid conditions. It might also be that most of the patients admitted to hospital were from average and por socioeconomic group who were unable to bear the cost of medicine & blood transfusion.

SUMMARY

Patients presenting with upper GI Bleeding in Government Mohan Kumaramangalam Medical College Hospital Salem, from December 2011 to November 2013, were studied.

Duodenal ulcer was the commonest cause of hematemesis and malena followed by oesophageal varices, gastric ulcer, erosive gastritis and reflux oesophagitis.

The peak incidence was among 35 to 45 years of age. Males were more predominant than female with male: female ratio.4:1

“O” blood group was commoner among the total cases of hematemesis and malena and it was also commoner among the duodenal ulcer bleeders.

Past history of epigastria pain was noted 15 (88%) and 6 (75%) cases of duodenal ulcer and gastric ulcer patients respectively. Past history of jaundice was obtained in 8 (80%) cases with oesophageal varices. Past history of hematemesis and malena was obtained in 2 (12%) & 3 (30%) cases of duodenal ulcer and oesophagcal varices.

Hematemesis ,malena and both hematemesis and malena were the presenting features of 30%,18%,and 52% of the patients .Among them 4(24%) patients of DU, 3 (38%) patients of GU & 4 (40%) patients of oesophageal varices

presented with hematemesis and 3 (17%) patients of DU 1 (13%) patients of GU & 2 (20%) patients of oesophageal varices presented with malena 10 (59%) patients of DU presented with hematemesis and malena.

Past history of epigastria pain was noted 15 (88%) and 6 (75%) cases of duodenal ulcer and gastric ulcer patients respectively. Past history of jaundice was obtained in 8 (80%) cases with oesophageal varices. Past history of hematemesis and malena was obtained in 2 (12%) & 3 (30%) cases of duodenal ulcer and oesophagcal varices.

Anemia, epigastric tenderness, splenomegaly were found in 74%, 60% and 18% cases respectively

In managing the cases with hematemesis and malena 65 patients (65%) were treated with 37 patients (74%) were treated with proton pump inhibitor (e.g. pantoprazole). Among the clinically suspected oesophageal varices patients two were treated with vasopressin and twelve were treated with octreotide, 9 were treated with banding.. Among the endoscopically diagnosed ulcer bleeders 10 were treated with sclerotherapy.

During the hospital stay, 1(5.9%), 1(2.5%), 3(30%) of duodenal ulcer, gastric ulcer and esophageal varices developed recurrent bleed.

CONCLUSION

Hematemesis and malena is a common medical emergency which needs immediate medical attention. Often it is very difficult to identify the underlying causes of hematemesis and malena.

Our study, done in Government Mohan Kumaramangalam Medical College Hospital, Salem; revealed results comparable with studies done in other parts of this country.

Peptic ulcer is the commonest cause of upper GI haemorrhage and the majority of the ulcers are located in the duodenum. Other common causes of hematemesis and malena are ruptured oesophageal varices and erosive gastritis. Sex and blood group distribution are almost similar with other studies conducted at home and abroad. But age distribution varies from country to country and it is a little bit lower in countries like us than those of western countries.

Endoscopic examination is the most important diagnostic tool in the diagnosis as well as management of upper GI haemorrhage.

Death due to hematemesis and malena is still a challenge for the investigators to modify treatment modalities to improve the prognosis.

Pitt falls

1.A large scale study is needed to reveal the actual situation regarding aetiology, prompt management to reach a favourable outcome in our locality.

2. Most of the patients presented very late after the development of symptoms, that could have influenced in tresult of our study

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ANNEXURE 1

PROFORMA

DISSERTATION ON UPPER GASTRO INTESTINAL BLEEDING

PROFORMA

Patient Details

Name: AGE: SEX: M / F
Address:

Occupation:
I.P. No:
DOA: DOD:

History

Hematemesis / Malena
Onset:
No. of Episodes:
H/O Bleeding from Gums / Nose:
H/O Dysphagia / Odynophagia:
Anorexia / weight loss:
H/O drug intake:

◯ NSAID ◯ ANTICOAGULANTS ◯ STEROIDS
◯ ASPIRIN ◯ ANY OTHERS:

H/O Bleeding PR:
H/O Jaundice:
H/O Passing clots in stool:
H/O forceful vomiting/retching:

Past History

H/O Peptic Ulcer:
Chronic Liver Disease:
Bleeding disorder:
Prev H/O:

◯ SHT ◯ Coronary Artery Disease:
◯ DM ◯ Prev H/O Surgeries:
◯ Bronchial Asthma:

Personal History

Diet:

◯ Smoker ◯ Alcoholic

Family H/O:

◯ Hematemesis ◯ G.I. Malignancy ◯ Bleeding disorders

General Examination

Built:

Nourishment:

◯ Consciousness	◯ Pallor	◯ Clubbing
◯ Oriented	◯ Icterus	◯ Lymphadenopathy
◯ Hydration	◯ Cyanosis	◯ Pedal Edema

Any signs of Liver Failure:

Oral Cavity

Skin Patechae/ Telangiectasias

VITALS

Pulse:	SPO_2:	Respiratory Rate:
Blood pressure:	Temperature:	Hourly Urine Output:

CVS:

RS:

CNS:

Ryles Tube aspirate

◯ Active Blood ◯ Altered Blood ◯ No Bleed

ATLS Shock Category

Stage: ◯ I ◯ II ◯ III ◯ IV

Per Abdomen

Per Rectal

WORK UP

Blood Grouping & Typing	:	Blood Sugar	:
Haemoglobin	:	Blood Urea	:
PCV	:	SR. Creatinine	:
Differential count	N: L:	Na+	:
Platelet Count	:	K+	:

URINE ANALYSIS

Albumin	:	RBCS	:
Casts	:	Bile Salts/Pigments	:

LIVER FUNCTION TESTS

Bilirubin – T	:	ALP	:
D	:	Protein – Albumin	:
SGOT	:	Globulin	:
SGPT	:	Total	:

COAGULATION PROFILE

BT	:	PT	:
CT	:	INR	:

SEROLOGY

HIV 1	:	HBS Ag	:
HIV 2	:	Anti HCV	:

ECG:
Echo:
X-Ray Chest PA View:
X-Ray Abdomen Erect View:

INITIAL MANAGEMENT

PROVISIONAL DIAGNOSIS

ULTRA SONOGRAM ABDOMEN

Liver:
Spleen:
Portal Vein:
Free Fluids

Any Other Findings:

CECT Abdomen (IF TAKEN)

UPPER GI ENDOSCOPY

FINAL DIAGNOSIS:

TREATMENT DETAILS

Condition on Discharge:

Follow Up:

ANNEXURE 2

PATIENT CONSENT FORM

PATIENT'S CONSENT FOR THE STUDY

I confirm that I have understood the purpose of surgical procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during surgical and post surgical procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relations to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third party or published unless as required under law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study for various surgical procedures and their outcomes. I understood everything written above in my vernacular language and has signed below with my conscious consent.

Time : ……

Date: …….....

Place: SALEM. **Signature/ thumb impression of the patient**

Signature of the investigator:……

ANNEXURE 3

MASTER CHART

PATIENT NAME	AGE	GENDER	I.P. NO	DOA	DOD	PLACE	SYMPTOMS			RISK FACTORS			VITALS		HEMATOCRIT		NGA	ATLS SHOCK SCORE	RAS	BLOOD GROUP	USG ABDOMEN	OGD SCOPY	MANAGED BY		REBLEED	HOSPITAL STAY	OUTCOME
							HEMATEMESIS	MALENA	JAUNDICE	SMOKING	NSAIDS	STEROIDS	PR	BP	HB%	PCV							PPI	OC	S / B / SUR			
PERIASAMY	44	M	22060	06/04/12	08/04/12	U	+	-	-	+	+	-	96	120/70	12.6	42	B	I	1	O	NORMAL	DUODENAL ULCER	+	-	-	3	D
ANNAMALAI	19	M	46633	25/04/12	26/04/12	R	+	+	-	+	-	-	90	110/70	10.8	32	B	I	1	B	CHOLELITHIASIS	DUODENAL ULCER	+	-	-	2	D
SYED IBRAHIM	38	M	22532	19/05/12	01/06/12	U	+	-	+	-	-	-	118	80/60	7	24	R	III	6	O	SPLENOMEGALY	VARICES	-	+	B	12	D
GOPAL	75	M	22640	12/06/12	15/6/12	U	+	+	-	-	+	-	102	106/76	11.6	39	C	II	3	O	NORMAL	GASTRIC EROSION	+	-	-	3	D
MARI	70	M	26150	25/06/12	09/07/12	R	+	+	+	+	-	-	122	70/50	6	22	R	III	10	B	CIRRHOSIS SPLENOMEGALY ASCITES	VARICES	+	+	B	14	E
RATHINAVEL	45	M	26182	08/07/12	14/07/12	R	+	+	-	+	+	+	108	100/70	10.8	34	R	II	5	O	NORMAL	DUODENAL ULCER	+	-	S	6	D
MARUTHAI	70	M	26192	28/07/12	03/07/12	R	+	-	-	+	+	-	102	110/70	9.2	30	R	II	5	AB	MILD SPLENOMEGALY	GASTRIC ULCER	+	+	S	5	D
PACHAIYAPPAN	37	M	26202	16/07/12	19/07/12	R	+	+	-	+	-	-	90	100/76	13.1	46	B	I	3	B	NORMAL	DUODENAL ULCER	+	-	-	3	D
GOKUL	32	M	88461	30/08/12	01/09/12	U	+	-	-	+	+	-	86	110/70	9.6	30	B	I	2	O	NORMAL	DUODENAL ULCER	+	-	-	2	D
DHANASEKAR	55	M	31592	10/10/12	22/10/12	R	+	-	+	-	-	-	106	90/70	8	28	R	III	7	O	SPLENOMEGALY ASCITES	VARICES	-	+	-	12	D

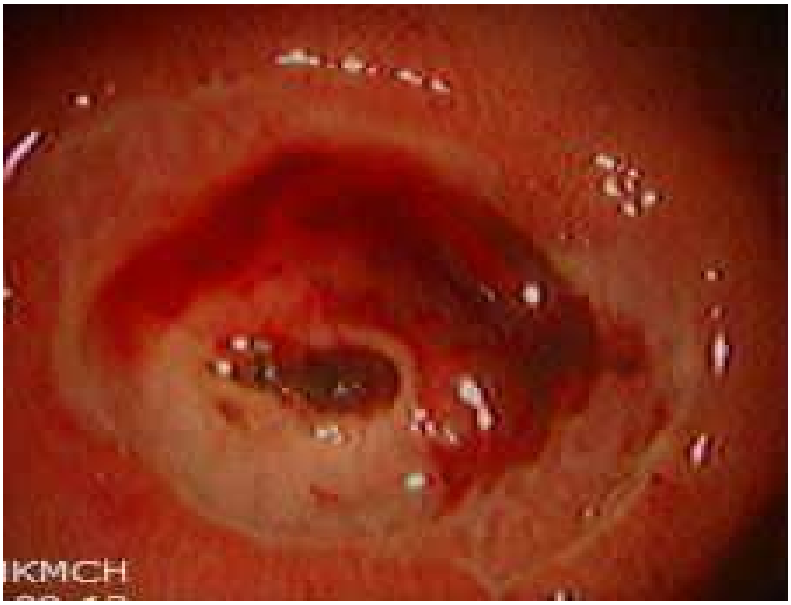
S.NO.	PATIENT NAME	AGE	GENDER	I.P. NO	DOA	DOD	PLACE	SYMPTOMS			RISK FACTORS			VITALS		HEMATOCRIT		NGA	ATLS SHOCK SCORE	RAS	BLOOD GROUP	USG ABDOMEN	OGD SCOPY	MANAGED BY		REBLEED	HOSPITAL STAY	OUTCOME
								HEMATEMESIS	MALENA	JAUNDICE	SMOKING	NSAIDS	STEROIDS	PR	BP	HB%	PCV							PPI	OC	S / B / SUR			
11	MURUGAN	50	M	8796	07/01/13	10/01/13	U	+	+	-	-	-	-	102	110/70	9.7	32	B	I	3	B	NORMAL	GASTRIC ULCER	+	-	-	-	3	D
12	SUBRAMANI	60	M	30393	30/12/13	02/12/13	U	+	+	-	+	-	-	80	120/70	8.6	30	R	I	4	AB	PARA AORTIC LN	DUODENAL ULCER	+	-	-	-	3	D
13	SAKKARAI	50	M	17402	18/01/13	20/01/13	R	+	-	-	+	-	-	88	120/70	12.8	40	C	I	0	B	NORMAL	NORMAL	+	-	-	-	2	D
14	PARAMASIVAM	52	M	43112	02/02/13	08/02/13	U	+	+	+	-	-	-	114	96/70	8.2	28	R	III	8	AB	CIRRHOSIS SPLENOMEGALY	VARICES	-	+	B	-	6	D
15	MOHAN	21	M	45218	05/02/13	11/02/13	R	+	+	-	+	+	-	102	90/60	10.7	34	R	III	5	O	HEMANGIOMA LIVER	DUODENAL ULCER ,CLOT	+	-	S	-	6	D
16	MADAIYAN SETHU	45	M	46126	04/03/13	09/03/13	R	+	-	-	-	-	-	98	100/60	9.4	32	B	III	5	AB	NORMAL	GASTRIC ULCER SPURT	+	-	S	-	4	D
17	ANANDAN	35	M	46128	04/03/13	08/03/13	R	+	+	-	+	-	+	88	110/70	8.2	29	R	I	3	O	NORMAL	DUODENAL ULCER	+	-	-	-	4	D
18	GANESAN	38	M	46132	04/03/13	07/03/13	R	-	+	-	+	+	-	82	130/80	12	38	C	I	1	O	NORMAL	GASTRIC EROSION	+	-	-	-	2	D
19	SOLOMAN	35	M	49950	18/03/13	23/03/13	U	+	+	-	+	-	-	114	94/62	10.8	34	R	III	5	AB	HORSE SHOE KIDNEY	DUODENAL ULCER	+	-	S	-	4	D
20	PONNUSAMY	68	M	7303	08/04/13	14/04/13	R	+	-	+	-	-	-	128	90/50	7.6	24	R	III	10	O	SPLENOMEGALY ASCITES	VARICES	-	+	B	-	6	D

S.NO.	PATIENT NAME	AGE	GENDER	I.P. NO	DOA	DOD	PLACE	SYMPTOMS			RISK FACTORS			VITALS		HEMATOCRIT		NGA	ATLS SHOCK SCORE	RAS	BLOOD GROUP	USG ABDOMEN	OGD SCOPY	MANAGED BY			REBLEED	HOSPITAL STAY	OUTCOME
								HEMATEMESIS	MALENA	JAUNDICE	SMOKING	ALCOHOL	STEROIDS	PR	BP	HB%	PCV							PPI	OC	S / B / SUR			
21	KUMAR	38	M	5198	08/04/13	12/04/13	R	+	+	-	+	-	+	86	110/70	7.2	26	B	I	3	O	NORMAL	GASTRIC ULCER	+	-	-	-	3	D
22	KADER BASHA	36	M	5238	10/04/13	16/04/13	R	+	-	-	+	-	-	122	82/56	6.5	18	R	III	5	A	NORMAL	DUODENAL ULCER ,CLOT	+	-	S	-	6	D
23	SADASIVAM	39	M	7050	28/04/13	02/05/13	U	+	+	-	+	-	-	102	100/64	8.6	22	R	III	4	O	SIMPLE CYST LIVER	DUODENAL ULCER,CLOT	+	-	S	-	4	D
24	LAKSHMI	55	F	7356	06/05/13	11/05/13	R	+	+	-	+	-	-	60	110/70	9.2	28	B	I	3	A	NORMAL	GASTRIC EROSION	+	-	-	-	4	D
25	TAJUDEEN	30	M	7128	08/05/13	13/05/13	R	+	-	-	+	-	-	106	110/70	11.4	34	C	II	1	O	NORMAL	MALLORY WEISS	+	-	-	-	4	D
26	MARIAMMAL	65	F	8125	18/05/13	30/05/13	R	-	+	-	+	-	-	92	116/72	10.6	33	R	II	5	O	ASCITES +	DUODENAL ULCER	+	-	-	-	12	D
27	ARUKKANI	60	F	9025	23/05/13	30/05/13	R	-	+	+	-	-	-	110	80/60	5	18	R	III	9	A	CELIAC NODES ASCITES	GASTRIC CARCINOMA	+	-	-	-	8	D
28	MURUGAN	39	M	20480	02/06/13	09/06/13	R	+	-	-	-	+	-	106	92/60	9.6	32	R	III	4	B	LIVER ABCESS	DUODENAL ULCER	+	-	S	-	6	D
29	SIVAGNANAM	56	M	23982	04/06/13	16/06/13	U	+	-	+	+	-	-	106	98/60	6.6	18	R	III	8	O	SPLENOMEGALY	VARICES	-	+	B	-	12	D
30	PANEER	57	M	24036	10/06/13	13/06/13	R	+	-	-	+	-	+	88	110/70	11.2	37	B	I	1	B	NORMAL	GASTRIC EROSION	+	-	-	-	2	D

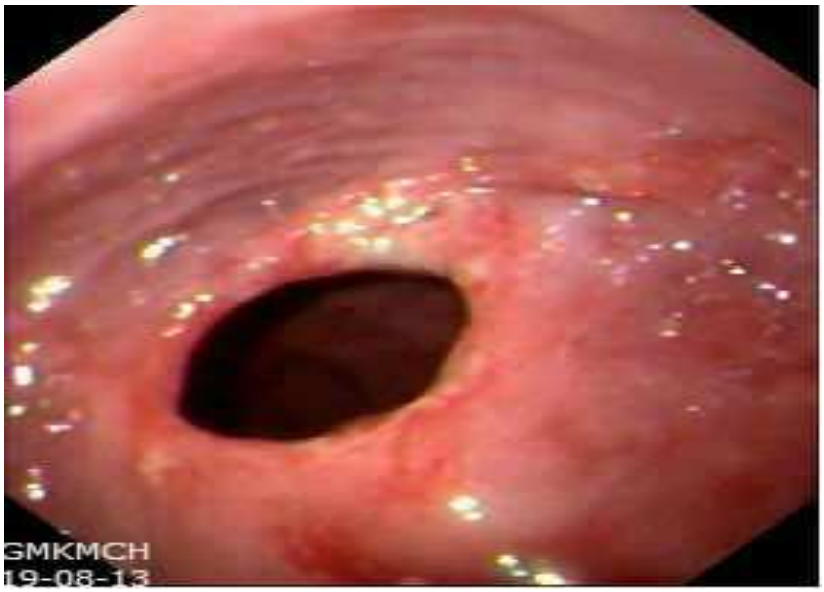
S.NO.	PATIENT NAME	AGE	GENDER	I.P. NO	DOA	DOD	PLACE	SYMPTOMS				RISK FACTORS			VITALS		HEMATOCRIT		NGA	ATLS SHOCK SCORE	RAS	BLOOD GROUP	USG ABDOMEN	OGD SCOPY	MANAGED BY		REBLEED	HOSPITAL STAY	OUTCOME
								HEMATEMESIS	MALENA	JAUNDICE	SMOKING	NSAIDS	STEROIDS	PR	BP	HB%	PCV							PPI	OC	S / B / SUR			
31	AMUDHAVALLI	68	F	25008	20/06/13	24/06/13	R	-	+	-	-	+	-	68	140/90	11.1	36	B	I	3	A	NORMAL	GASTRIC ULCER	+	-	-	-	4	R
32	GOVINDARAJ	28	M	24050	12/06/13	18/06/13	R	+	+	-	-	-	+	92	140/80	9.6	34	R	I	4	B	NORMAL	DUODENAL ULCER	+	-	-	+	6	R
33	DHANAPAL	45	M	25014	22/06/13	30/06/13	U	-	+	-	-	+	-	86	130/80	8.8	34	B	I	3	O	CHOLELITHIASIS	DUODENAL ULCER	+	-	-	-	8	R
34	SHAJAHAN	50	M	25866	03/07/13	09/07/13	U	+	-	-	+	-	-	90	100/70	7.8	30	R	I	3	B	FATTY LIVER	GASTRIC ULCER SPURT	+	-	-	+	6	R
35	ANNADURAI	65	M	25802	06/07/13	17/07/13	U	+	+	-	+	-	-	92	90/60	5.5	16	R	III	7	AB	NORMAL	GASTRIC CARCINOMA	-	-	SUR	-	10	R
36	SRINIVASAN	32	M	48768	09/08/13	12/08/13	U	+	+	-	+	-	-	102	110/70	8.0	28	R	II	4	B	NORMAL	TELAN	+	-	S	-	3	R
37	BAKKIYAM	60	F	48802	11/08/13	15/08/13	U	-	+	+	-	+	+	88	110/70	7.4	26	R	I	4	A	FATTY LIVER	VARICES	-	+	B	-	4	R
38	MANIMEGHALAI	22	F	49021	18/08/13	22/08/13	R	+	-	-	-	-	-	86	100/70	11.2	36	B	I	1	O	NORMAL	ESOPHAGITIS	+	-	-	-	3	R
39	GANESAN	58	M	43468	08/09/13	18/09/13	R	-	+	+	+	-	-	102	90/70	5.4	16	R	III	9	O	SPLEEN + ASCITES P.V.- 13	VARICES	+	+	SB	+	10	R
40	KANDASAMY	72	M	32412	06/09/13	08/09/13	R	+	+	-	+	+	+	80	140/90	13.5	44	C	I	3	O	HEMANGIOMA LIVER	NORMAL	+	-	-	-	2	R

S.NO.	PATIENT NAME	AGE	GENDER	I.P. NO	DOA	DOD	PLACE	SYMPTOMS				RISK FACTORS			VITALS		HEMATOCRIT		NGA	ATLS SHOCK SCORE	RAS	BLOOD GROUP	USG ABDOMEN	OGD SCOPY	MANAGED BY			REBLEED	HOSPITAL STAY	OUTCOME
								HEMATEMESIS	MALENA	JAUNDICE	SMOKING	ALCOHOLISM	STEROIDS	PR	BP	HB%	PCV							VARICES	PPI	OC	S / B / SUR			
41	HUSSAIN	50	M	37688	16/09/13	23/09/13	R	+	+	+	+	-	-	116	96/70	7.1	28	R	III	7	B	SPLENOMEGALY CIRRHOSIS	DUODENAL ULCER SPURTING VESSEL	-	+	B	-	6	D
42	PALANIVEL	45	M	39334	18/09/13	21/09/13	R	+	+	-	-	-	-	126	70/50	5.2	16	R	III	6	AB	NORMAL	GASTRIC ULCER	+	+	S	-	3	E
43	THANGAMMAL	54	F	41256	23/09/13	26/09/13	R	+	+	-	-	+	-	66	140/90	8.6	30	B	I	3	O	FIBROID UTERUS	GASTRIC ULCER	+	-	-	-	3	D
44	KUMAR	23	M	42223	03/10/13	05/10/13	R	+	+	-	+	+	-	86	120/70	14.6	46	C	I	1	B	R HUN	GASTRIC EROSION	+	-	-	-	2	D
45	WILSON	43	M	42382	10/10/13	14/10/13	U	+	+	-	+	-	-	104	100/70	11.6	40	B	I	4	O	NORMAL	GASTRIC ULCER CLOT	+	-	S	-	4	D
46	SAROJA	46	F	41432	28/09/13	02/10/13	R	-	+	-	-	+	-	90	100/70	10.9	39	R	II	4	AB	NORMAL	DUODENAL ULCER	+	-	-	-	4	D
47	VELAMMAL	49	F	42460	13/10/13	17/10/13	R	+	+	-	-	-	-	96	110/70	11.8	38	B	I	1	B	NORMAL	ESOPHAGITIS	+	-	-	-	4	D
48	SYED AKRAR	65	M	42532	27/10/13	06/11/13	U	-	+	+	+	-	-	90	100/70	7.2	28	R	I	6	AB	ASCITES	ESOPHAGEAL CARCINOMA	+	-	SUR	-	8	D
49	PERIASAMY		M	43216	03/11/13	07/11/13	U	+	+	-	+	-	-	82	110/70	15.0	43	C	I	3	O	GB SLUDGE	NORMAL	+	-	-	-	4	D
50	PONNUTHAYEE	48	F	44316	02/11/13	12/11/13	R	+	+	+	-	-	-	110	80/60	8.8	34	R	III	7	B	ASCITES CIRRHOSIS	VARICES	+	+	B	-	10	D

PLATE 1



BLEEDING DUODENAL ULCER



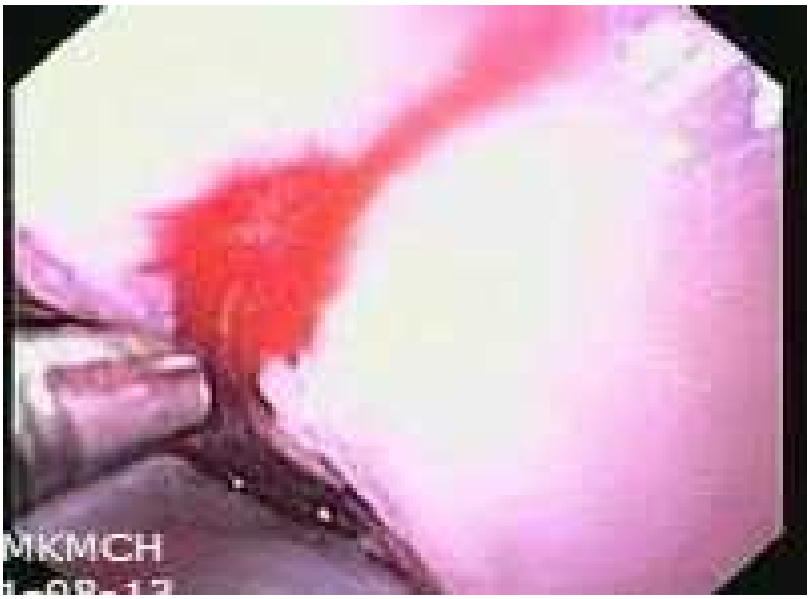
ESOPHAGITIS

PLATE 2

ESOPHAGEAL VARICES

CARCINOMA STOMACH

PLATE 3



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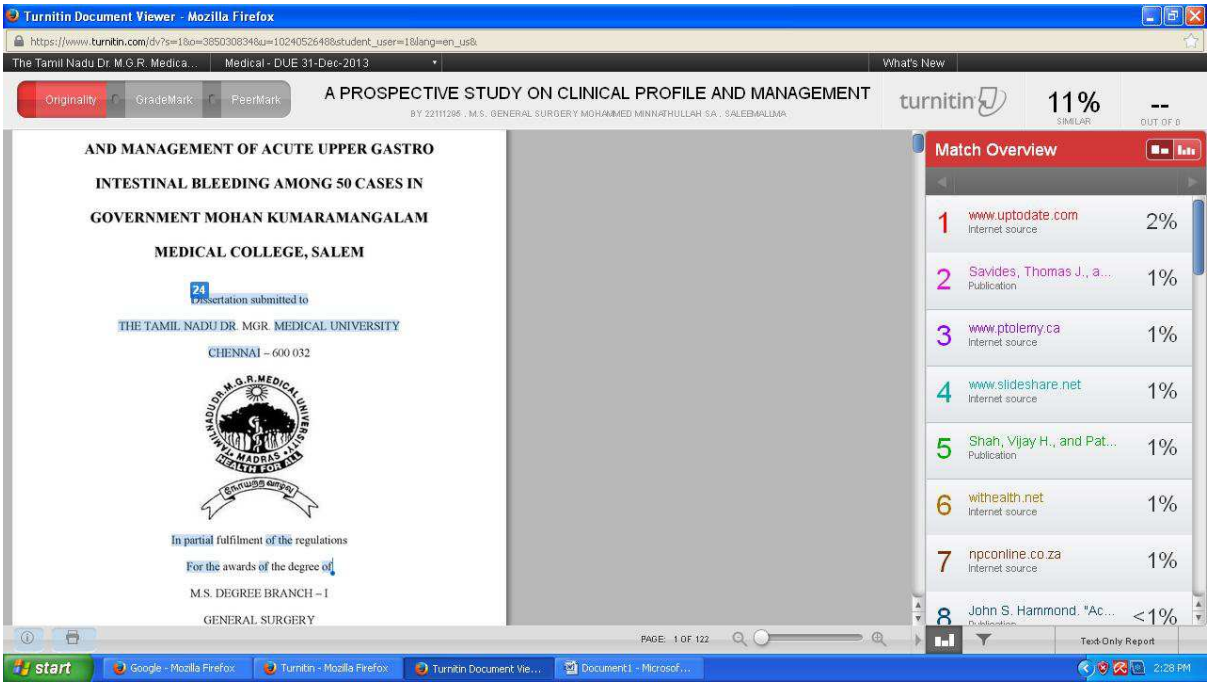
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MEDICAL COLLEGE, SALEM
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